THE IMPACT OF URBANISATION AND INDUSTRIALISATION IN MEDIEVAL AND POST-MEDIEVAL BRITAIN

An assessment of the morbidity and mortality of non-adult skeletons from the cemeteries of two urban and two rural sites in England (AD 850-1859)

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ABSTRACT

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THE IMPACT OF URBANISATION AND INDUSTRIALISATION IN MEDIEVAL AND POST-MEDIEVAL BRITAIN

AN ASSESSMENT OF THE MORBIDITY AND MORTALITY OF NON-ADULT SKELETONS FROM THE CEMETERIES OF TWO URBAN AND TWO RURAL SITES IN ENGLAND (AD 850-1859)

KEYWORDS: MEDIEVAL, NON-ADULTS, URBAN-RURAL, STRESS, GROWTH, ENVIRONMENT, ENGLAND.

This study compares the morbidity and mortality of non-adults interred in urban and rural cemeteries between AD 850-1859. It was hypothesised that the development of urbanisation and industrialisation, with subsequent overcrowding and environmental pollution, would result in a decline in human health in the urban groups. This would be evident in lower mean ages at death, retarded growth and higher rates of childhood stress and chronic infection in the children living in the urbanised environments.

Non-adult skeletons were examined from Raunds Furnells in Northamptonshire (Anglo-Saxon), St. Helen-on-the-Walls in York (later medieval, urban), Wharram Percy in Yorkshire (later medieval, rural) and from the crypt of Christ Church Spitalfields, in London (AD 1729-1859). The results showed that it was industrialisation, rather than urbanisation that was most detrimental to child health. Weaning ages declined from two years in the Anglo-Saxon period to one year in the eighteenth and nineteenth century. Industrialisation was characterised by a lower mean age at death, growth retardation and an increase in the prevalence of rickets and scurvy. Although higher rates of dental disease and maternal stress were apparent in the urbanised samples, respiratory diseases were more common in the rural areas. Growth profiles suggested that environmental factors were similar in the urban and rural communities in the later medieval period. However, there was evidence that employment had a detrimental effect on the health of later medieval apprentices.

This study demonstrates the importance of non-adult remains in addressing issues of health and adaptation in the past and, the validity of using skeletal material to measure environmental stress.

CONTENTS

CHAPTER THREE: SKELETAL INDICATORS OF STRESS AND ADAPTATION

CHAPTER FOUR: SKELETAL CHANGES OF INFECTION AND METABOLIC DISEASE

CHAPTER FIVE: MATERIALS: HISTORICAL AND ARCHAEOLOGICAL BACKGROUND TO THE SITES

CHAPTER SIX: METHODS

SUPPORTING PUBLICATIONS

Lewis, ME, and Roberts, CA (1997) Growing Pains: The Interpretation of Stress Indicators. *International Journal of Osteoarchaeology* 7: 581-586.

In Press:

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CHAPTER ONE

INTRODUCTION

'The pattern of disease or injury that affects any group of people is never a matter of chance. It is invariably the expression of stresses and strains to which they were exposed, a response to everything in their environment and behaviour. It reflects their genetic inheritance, the climate in which they lived... and the animals or plants that shared their homeland. It is influenced by their daily occupations, their... diet, choice of dwelling... clothes and their social structure.'

(Wells, 1964: *17*)

1.1 BACKGROUND TO RESEARCH

1.1.1 NON-ADULT SKELETONS AS MEASURES OF STRESS AND ADAPTATION

The health and survival of the children from any given community is believed to represent the most demographically variable and sensitive index of biocultural change (Roth, 1992; Van Gerven and Armelagos, 1983). Patterns of infant and child mortality have been shown to have a profound effect on the crude death rates of a population and, when coupled with evidence of childhood morbidity, have become accepted as a measure of population fitness (Mensforth *et al.*, 1978). In other words, the health and survival of the offspring indicates the level to which a population has adapted to the environment in which they live. Roth (1992) described childhood as the most sensitive portion of the human life cycle, and Saunders and co-workers stated that infant mortality is '*...generally considered to be an important public health index for assessing the sanitary and social conditions that surrounded an infant in life*' (Saunders *et al.*, 1995: *71*). Nevertheless, until recently the study of non-adult remains in biological anthropology has been limited. Small sample sizes, poor preservation and the inability to assign a sex to non-adult skeletons, has led researchers to concentrate on adult skeletal material for insights into population health and adaptation.

The study of 'growth' rather than a static adult stature means that the age at which children fall behind or recover their growth trajectory can be examined. In addition, skeletal and dental markers of stress develop during childhood, and most proceed to remodel as the individual gets older. Therefore, the true prevalence of lesions such as cribra orbitalia or Harris lines can be more accurately assessed in younger individuals. The precise age at which these lesions developed can

help us answer questions about the nature of stress and survival (Goodman, 1993; Wood *et al.*, 1992). However, studies of purely non-adult samples are limited in both Europe and the USA. In Britain, studies of large groups of non-adults to answer particular archaeological questions are rare, and usually concentrate on a specific sample rather than a comparison of children from different populations and periods.

Nevertheless, the children in the archaeological sample represent the '*non-survivors*' from any given population, and their pattern of growth or frequency of lesions might not reflect that of the children that went on to survive into adulthood (Saunders and Hoppa, 1993; Wood *et al.*, 1992). The early death of these individuals provides other challenges in the study of non-adult palaeopathology. Chronic diseases need time to develop on the skeleton, but the children that enter the archaeological record have usually died in the acute stages of these diseases before the skeleton has had time to respond. At the present time, studies that concentrate on non-adult material are hindered by the inability to make reliable sex estimations, due to absence of the secondary sexual characteristics evident on the adult skull and pelvis. Although sexual dimorphism has been identified *in utero*, there is still a disagreement about the validity of identifying morphological traits indicative of sex in the non-adult skeleton (Halcomb and Konigsberg, 1995; Schutkowski, 1993). However, the application of DNA analysis in determining the sex of non-adult skeletal material has begun to be put to use (Colson *et al.*, 1997), and holds promise for the future. However, the time and money that would be needed to sex all the non-adults in a sample would restrict any major research in this area.

1.1.2 The development of Comparative Urban-rural Studies

The interaction of humans with their environment has been an issue in the anthropological and medical literature for many decades. More recently, biological anthropologists have begun to examine how humans have adapted to their changing environment in the past, using both biological and cultural data, a trend known as the '*biocultural approach*'. In palaeopathology, in particular, this new theoretical paradigm has resulted in a move away from the study of individual skeletons and diseases, to the analysis of population health using multiple indicators of stress.

Investigations into the health of past populations making the transition from a hunter-gatherer to an agricultural subsistence have received much attention in biological anthropology. The majority

of these studies come from North America and they suggest that sedentism and reliance on a less diverse food supply was detrimental to the health of early farmers (Cohen and Armelagos, 1984; Gilbert and Mielke, 1985; Johansson and Horowitz, 1986; Swedlund and Armelagos, 1990). However, little has been done to evaluate and compare the health status of past populations living in the rural, urban and industrial centres of Europe or North America, using human skeletal remains. It is believed that differences in , for example, housing, diet, sanitation, population density and trade relations would have exposed the inhabitants to a diverse range of environmental pathogens, leading to contrasting patterns of morbidity and mortality in urban and rural settlements.

Theoretical models examining the evolution of the host-pathogen relationship between humans and their environment have shown that, with the development of urban centres, acute population dependent diseases flourished, with large numbers of potential hosts being replenished by rural migrants, who were attracted to those urban areas (Cockburn, 1971). This migration not only provided new 'virgin' hosts but also led to new diseases being introduced into this lucrative environment (McNeill, 1976; 1979; Luckin and Mooney, 1997), with diseases such as cholera, typhoid, typhus, smallpox and the plague thriving in overcrowded and unsanitary conditions. This change in the pattern of disease has been illustrated in the historical literature, and to some extent in the skeletal material from Britain, with chronic infectious diseases such as leprosy, treponemal disease and tuberculosis becoming prevalent with the development of urbanisation in the later medieval period (Manchester, 1992).

Fully industrialised centres, such as those in the eighteenth and nineteenth centuries in England, were even more densely populated and particularly dependent on external food supplies from the countryside and through trade. The gap between the social classes became more distinct at this time, and industrial advances resulted in an increase in environmental pollutants (Storey, 1992). Historical data records a rise in complaints about air pollution starting in the medieval period (Brimblecombe, 1975; 1976; 1978a; 1978b; Brimblecombe and Bowler, 1992), with growth retardation (Meredith, 1982) and higher rates of infant mortality in the urban environment (Williams and Galley, 1995). Historians have also recorded an increase in the prevalence of rickets and scurvy with industrialisation (Fildes, 1986b), and recent access to post-medieval material from modern crypt clearances in England has provided the biological anthropologist with the potential to analyse the health of populations in industrial environments. Today, most of the

physical problems associated with an urban lifestyle have been conquered in the Western world, only to be replaced by psychological stress, resulting in a greater prevalence of mental illness, depression, violence and alcoholism (Boyden, 1972; Russell and Russell, 1970).

One of the first attempts to assess the impact of urbanisation using skeletal material was by Wells in 1977. He examined the prevalence of maxillary sinusitis from the Bronze Age through to the later medieval period in Britain, and found an increase in the condition with time. In Central America, Storey (1988) examined the health of infants and children from two contrasting sites in Mexico; Teotihuacan, a low status, densely populated urban centre and, the high-status, low-density site of Copan. The increase in morbidity and greater mortality of the Teotihuacan children was taken as indicative of the worsening effects of urbanisation. After a hiatus in this area of research, studies into the health of populations living in urban environments gradually increased, and this was reflected in the publication of '*Death in Towns*' edited by Bassett (1992). This book drew together evidence for the health of populations living in urban centres from the Roman to later medieval periods in Britain. The various authors employed archaeological, historical and skeletal evidence to examine the nature of urbanisation within these time frames. However, none of the skeletal studies contained in this book set out to compare the health of the urban inhabitants with their rural counterparts but, instead, discussed the effects of urban living on human health in isolation. However, in 1994 Brothwell began to redress this situation when he carried out a brief review of urban and rural cemetery sites in and around medieval York. He compared the cranial metrical data, stature and prevalence of cribra orbitalia (a lesion thought to be indicative of iron deficiency anaemia) using published data, but his results were inconclusive and were not developed any further. A similar attempt to assess health in contrasting settlements was carried out in Spain by De la Rúa and co-workers (1995). They analysed the prevalence of skeletal markers in urban and industrialised centres, compared to agricultural and transitional populations, from published sites all over the world and found an increase in skeletal and dental markers of stress with urbanisation.

In 1995, Lewis and colleagues developed the work carried out by Wells (1977) and compared the prevalence of maxillary sinusitis in an urban and a rural community from later medieval Yorkshire. They found a greater prevalence of the condition in the urban group. More recently, Judd and Roberts (1999) examined the patterns of trauma in urban and rural groups from Britain and concluded that an agricultural lifestyle contained more physical hazards than an urban one.

Both of these later studies have suggested a new dimension in the analysis of urban and rural environments, and have illustrated, not that a rural lifestyle was necessarily better for our ancestors, but that the threats to human health were different.

1.1.3 Skeletal Measures of Environmental Stress

Individuals displaying pathological lesions and indicators of stress in any given skeletal population have traditionally been thought to represent the most disadvantaged individuals from that society. These individuals may have had a depressed immune system, been malnourished and probably lived in an overcrowded, unhygienic environment that would have increased their risk of exposure to a greater number of pathogenic organisms. In contrast, those without lesions are thought to represent individuals whom, perhaps due to nutritional and cultural advantages, survived with fewer episodes of stress until their time of death.

In 1992, Wood and co-authors challenged these preconceptions and warned, in their paper entitled '*The Osteological Paradox*', that any attempt by osteologists to reconstruct the health patterns of past populations from skeletal remains was paradoxical, as these data only provide information about the morbidity and mortality rates of '*non-survivors*'. The frequencies of disease in a skeletal assemblage would be higher than for the surviving population from which they were derived and therefore, both demographic and palaeopathological methods are fundamentally flawed. More importantly, Wood and colleagues (1992) argued that those individuals with dental and skeletal markers of stress, often seen as the '*disadvantaged,*' actually had the ability to survive episodes of disease and recover from them. Without a relatively strong immune system they would have died before any lesions could appear on the skeleton. Therefore, they argued, individuals without recognisable lesions on the skeleton may have been too weak to survive a period of stress and were actually the more 'disadvantaged' members of the community. However, current methods would not be able to distinguish this group from the most '*advantaged*' sections of the community, who never experienced stress at all, and therefore, had no markers of stress on their skeletons.

These arguments were counteracted by Goodman's contention that the age of the individuals at the time of their death would provide a measure of their ability to survive. An early death would point to a lack of adaptation (Goodman, 1993). Central to this argument is the need to be able to age individuals accurately, something that has been a matter of contention for the adult skeletal

material for many years (Bocquet-Appel and Masset, 1982; Buikstra and Konigsberg, 1985; Jackes, 1992). However, age estimates of non-adults are determined using age specific markers of growth, mainly of the dentition. The timing and outcome of these events is thought to be linked to a genetic blueprint, unlike the more variable degenerative age determinants of adulthood (Rallison, 1986). For this reason, non-adult skeletons allow for a more precise skeletal age estimation than is possible for adults.

1.2 Research Aims

This research developed out of the need to address the issues surrounding the potential impact of urbanisation and industrialisation on human health. This was achieved by investigating the changes in morbidity and mortality of non-adult individuals (up to 17 years of age) from four contrasting sites in England dating from AD 850 to 1859, and spanning the period of urban and industrial development. The hypothesis tested is that, as Britain became more industrialised, the health of the inhabitants deteriorated. Any failure in a population's ability to adapt to their environment should be evident in the non-adult skeletons in the form of a lower mean age-at-death, retarded growth, higher levels of stress and a greater prevalence of metabolic and infectious disease.

The sites used in this study were chosen for their geographical location, large non-adult sample size and contrasting settlement character. Raunds Furnells in Northamptonshire is the earliest of the settlements, with a cemetery dating to between the tenth and twelfth centuries. The inhabitants of this Anglo-Saxon settlement would have practised subsistence agriculture. The parish of St. Helen-on-the-Walls, based in pre-industrial York, dates from the eleventh to sixteenth centuries and was contemporaneous with Wharram Percy, a deserted medieval village in the Yorkshire Wolds (c. AD 900-1500). Although the inhabitants of Wharram Percy would have been living in a rural environment, they would also have had contact with the urban centre of York. Finally, non-adults excavated from the crypt of Christ Church Spitalfields, in London (AD 1729-1859) were chosen to represent the post-medieval sample. Archaeological, historical, social anthropological and clinical data was used to assess the nature of the urban and rural environment and potential health problems its inhabitants may have endured. The objectives of this research were:

- To assess differences in the levels of morbidity and mortality in non-adults from urban and rural environments
- To assess the impact of urbanisation and industrialisation on human health in medieval and post-medieval England
- To examine the types of evidence for morbidity that can be observed on non-adult skeletons
- To evaluate the use of skeletal and dental indicators of stress as measures of environmental change
- To assess what factors in the urban and rural environments may be contributing to any differences between the samples.

1.3 STRUCTURE

The following chapters discuss in detail previous research into the nature of urban and rural environments, using data from the archaeological, medical, historical and social anthropological literature. Chapter Two examines the archaeological and environmental evidence for living conditions in the town and countryside in the light of host-pathogen relationships. The development of urbanisation and industrialisation in Britain is also examined and changing attitudes towards hygiene, environmental pollution and diet are explored. The chapter finishes with a review of modern medical research in urban and rural communities in Britain, the USA and developing countries, and outlines the health measures currently used in modern medical and historical research to evaluate the impact of the urban environment on human well being.

Chapter Three presents the biological concepts of stress and adaptation used in modern clinical literature and, examines how these theories have been integrated into studies of past populations. This chapter also introduces the many skeletal indicators of stress employed in biological anthropology and discusses their origins, development, potential and limitations. The following chapter continues this line of enquiry and examines the evidence for infectious and metabolic diseases that can be identified in non-adult skeletal material. The historical and palaeopathological backgrounds of these conditions are discussed with particular reference to their occurrence in urban and industrial populations.

The archaeological and historical backgrounds to the sites under study are contained in Chapter Five, and includes an evaluation of the non-adult samples to assess whether infants (under one year) and children are well represented in each site. Chapter Six outlines the methods employed in this research, and the appendices contain photographs and details of the various grading schemes used, as well as the raw data from the skeletal analysis.

The Results Chapter presents the evidence for infant and child mortality, compares the growth profiles of the urban and rural children, examines the influence of various indicators of stress on growth and longevity, and assesses the prevalence of infectious and metabolic diseases in the sites under study. The final chapters (Chapters Eight and Nine) discuss these results in the light of previous research, and assess the effectiveness of both non-adult material and skeletal markers of stress in measuring environmental change.

CHAPTER TWO

HEALTH IN THE URBAN AND RURAL ENVIRONMENT

2.1 INTRODUCTION

Today, issues of health in urban-rural environments in the developed and developing world are commonly measured by the growth and development of children, fertility and demography of the population, levels of morbidity and mortality, and by measuring the health of the rural migrant population compared to rural non-migrants (*sedents*). It is argued that any detrimental effects of the urban environment will be evident in reduced fertility, retarded growth and development, greater disease loads and higher rates of mortality (Bogin, 1988b).

When attempting to assess differences in urban and rural environments, both in the past and today, the nature of urbanisation, cultural factors and the social status of the group under study need to be taken into account (Tanner and Eveleth, 1976). In general, modern urban communities benefit from access to a wide variety of foods, greater wealth and more sophisticated medical facilities than are available to the rural inhabitants. Conversely, people in rural areas have less exposure to pollution and take more exercise (*ibid.*, 1976). Evidence from the developing world suggests that urbanisation attracts large numbers of migrants that travel to the city from rural areas to find work. The lack of adequate housing and sewage disposal provides an ideal environment for waterborne diseases and childhood infections such as measles, whooping cough and dysentery, and exposes new migrants to pathogens and environmental conditions never experienced before. Inadequate pollution control results in serious occupational hazards and mental health problems, and maternal deaths and sexually transmitted diseases are a growing problem (Susser, 1987; Atkinson, 1993).

2.1.1 DEFINITIONS OF 'URBAN AND 'RURAL'

Today, many researchers use population density as a measure of urbanisation, where 'rural' is defined as a settlement with less than one thousand inhabitants, or where the majority are employed in subsistence agriculture (Williams and Galley, 1995). However, Watt and co-workers (1994) argue that this is not an appropriate measure, as many commercial inner city areas have less than one thousand inhabitants, with the majority residing in the suburbs. Others argue that

social complexity, function, and the number and variety of services provided by the town is a better measure of urbanity (Grove, 1972; Smith, 1972).

Sorokin and Zimmerman (1970) provided a table outlining the fundamental characteristics of urban and rural environments (Table 2.1). They describe rural settlements as more homogenous and closer to the natural environment and, despite being highly mobile, they have a close kinship network, compared to urban areas where population density, heterogeneity, isolation from nature and transient relationships dominate. Here, the factors outlined by Sorokin and Zimmerman (1970) are adhered to and an *urban* settlement is defined as a large, dense population with a distinct and diverse range of economic functions, as well as having evidence for an established administration, politics and religion (Ottaway, 1992). *Rural* communities are understood to have smaller population numbers with the inhabitants being mainly involved in agricultural activities (Williams and Galley, 1995).

The following sections will discuss the different environmental factors that existed in urban and rural settlements in medieval and post-medieval communities, during the periods from which the study samples are derived (AD 850 to 1859). Evidence for urban-rural health from both historical records and modern developing countries will be discussed in relation to levels of hygiene, host-parasite relationships, diet, air pollution, climate and mortality, with particular emphasis on the diet, health and mortality of infants and children.

There are two aspects of the environment which have an important bearing on disease, the general environment, including the climate, technology, the water supply and the disposal of sewage and waste, and the internal or micro-environment comprising of individual domestic arrangements for food storage and preparation, waste disposal and hygiene (Luckin, 1980). All of these factors have an effect on the development of the host-parasite relationship and, before specific details about the nature of the urban and rural environment and its effect on health can be discussed, the theories behind the host-parasite relationship need to be explained.

Table 2.1 Fundamental Characteristics of Rural and Urban Environments. After Sorokin and Zimmerman, 1970: 75.

	Rural	**Urban**
Occupation	Totality of cultivators and their families. In the community there are usually a few representatives of several non-agricultural pursuits.	Totality of people engaged principally in manufacturing, mechanical pursuits, trade, commerce, professions, governing and other non-agricultural occupations.
Environment	Predominance of nature over anthropo-social environment. Direct relationship to nature.	Greater isolation from nature. Predominance of human environment over natural. Poorer air. Stone and iron.
Size of Community	Open farms or small communities. Agriculturalism and size of the community are negatively correlated.	As a rule, in the same country in the same period, the size of the urban community is much larger than the rural community. In other words, urbanity and size of community are positively correlated.
Population Density	In the same county and at the same period, the density is lower than in the urban community. Agriculturalism and density are negatively correlated.	Greater than in the rural community. Urbanity and density are positively correlated.
Heterogeneity and Homogeneity of Population	Compared to urban populations, rural groups are more homogenous in racial and psychosocial traits.	More heterogeneous than rural communities. Urbanity and heterogeneity are positively correlated.
Social Differentiation and Stratification	Rural differentiation and stratification are less than urban.	Differentiation and stratification show positive correlation with urbanity.
Mobility	Territorial, occupational and other forms of social mobility of the population are comparatively less intensive. Normally the migration current carries more individuals from the country to the city.	More intensive. Urbanity and mobility are positively correlated. Only in periods of social catastrophe is the migration from the city to the country greater than from the country to the city.
System of Interaction	Less numerous contacts per person. Narrower area for the interaction system, and its members are the whole aggregate. More prominent part is occupied by primary contacts. Predominance of, or personal and relatively durable, relationships. Comparative simplicity and sincerity of relations. Interacted with as a 'person'.	More numerous contacts. Wider area of interaction system per human and per aggregate. Predominance of secondary contacts and impersonal, casual and short-lived relationships. Greater complexity, manifoldness, superficiality and standardised formality in relations. Interacted with as a 'number' and 'address'.

2.2 HOST-PARASITE RELATIONSHIPS

2.2.1 IMMUNITY AND EXPOSURE

Immunity is concerned with the recognition and destruction of foreign or 'non-self' material that enters the body. Resistance to infection from these organisms is *innate* (natural), *passive* or *acquired* (Playfair, 1992). Innate immunity does not require previous exposure to a pathogen, is genetically based and aided by physical barriers such as the skin, phagocytic cells, and certain chemical substances or enzymes. Passive immunity in newborns involves the transplacental transmission of antibodies from the mother to her unborn child and, after birth, through the breastmilk. Acquired immunity results from exposure to a specific organism, its elimination, and recovery from disease. Subsequent exposure to that organism leads to a more efficient and effective immune system, as the body responds using a specific memory for the organism, producing tailor-made antibodies to fight the infection, making the individual exempt from further attacks (Playfair, 1992; Weir and Stewart, 1997).

Exposure to a pathogen (virus, fungus, single-celled organism or bacterium) does not automatically result in infection. Many factors, including the mode of transmission, abundance of the pathogen, and physical factors such as climate, temperature, humidity and sunlight have an effect on the pattern of disease and may hinder or aid disease transfer (Howe, 1982). Once infected, the clinical signs of disease do not always develop and rely on the infectivity, pathogenicity and virulence of the parasite, as well as host factors such as their genetic and nutritional status, age, sex, stress levels and previous exposure. Environmental factors including population density, levels of hygiene and access to medical care influence whether the host is susceptible to exposure and infection (Mascie-Taylor, 1993). Host and parasite have evolved together with a trend from an acute to a more chronic disease and most of the diseases that affect the skeleton are the result of a long evolutionary process in the relationship between host and parasite. Initially exposure of a population to a pathogen will result in an epidemic, where an abundance of new hosts allow for the rapid spread of the disease and high mortality. With prolonged exposure, the population develops an increased resistance to the pathogen with a smaller number of new hosts being exposed and acquiring immunity to the disease, which becomes endemic (Ikwueke, 1984).

In biological anthropology, changing patterns of disease are studied using ecological or evolutionary models, which examine the interaction between the pathogen and host within specific environments. The biocultural approach has been developed to investigate how cultural systems

can affect disease processes, and to measure the influence of disease on cultural change (Armelagos, 1990; Fenner, 1971; Larsen, 1997). For example, Cockburn (1977) argued that in small hunter-gatherer communities, with few susceptible people, the success of a pathogen relied on its ability to survive until new hosts appeared, that is, they lived in a commensal relationship with the host, causing chronic disease without it leading to death. More acute diseases such as measles, relied on large numbers of prospective hosts, provided by the expanding agricultural and urban communities (Cockburn, 1971). McNeill (1976; 1979) and Luckin and Mooney (1997) have illustrated how migration leads to an increase in population and the introduction of new diseases, while the migratory population, as virgin hosts, are susceptible to new environmental pathogens and to the stress of social change.

2.2.2 The Rural Environment

An increase in agricultural intensification had a detrimental effect on human health in many societies throughout the world (Cohen and Armelagos, 1984). The transition from a hunter-gatherer to an agricultural subsistence in the Neolithic period in Europe, for example, removed some of the potential threats to human health, such as serious injury during hunting or exposure to zoonoses during the butchery of wild animals, but led to the introduction of new health hazards (Boyden, 1972). The development of agriculture meant people were dependent on a limited and less varied food supply, and crop failure could lead to pestilence and famine. Sedentism led to greater local environmental contamination, where permanent housing and attachment to land meant that people were no longer free to leave an area before pathogens could become established. An increase in population size resulted in closer contact with other humans and livestock, making the agricultural settlement an ideal place for disease transmission, as infections spread from person-to-person, or animal-to-person, via the respiratory tract (Cohen, 1989; Manchester, 1992; Roberts and Manchester, 1995). The domesticated cow, pig, sheep, cat and dog were accompanied by the rat, mouse, tick, flea and mosquito which acted as vectors for disease (Cockburn, 1971). This closer contact of humans with livestock provided the opportunity for zoonoses to adapt to a human host and new diseases began to emerge (*ibid.*, 1971). For example, measles is believed to have originated from the distemper virus in dogs (Mims, 1980) and tuberculosis probably developed from the bovine form of the disease, through ingestion of infected meat and milk, and respiratory transmission (Roberts and Manchester, 1995). Respiratory infections would have been spread in the confines of houses and during the winter, the burning of

fires would have exposed the inhabitants to the detrimental affects of smoke. Contact with the milk, meat, skin and hair of domestic animals would also have exposed farmers to anthrax and brucellosis (Armelagos, 1990).

Cultivation of new tracts of land for agricultural exploitation exposed people to new vectors and their pathogens. For example, mosquitoes would have had little contact with humans before deforestation and swamp drainage, which allowed for the transmission of yellow fever, dengue, scrub typhus and malaria (Cohen, 1989; McGrath, 1992). Contact with the soil exposed people to fungal diseases such as those caused by *Aspergillus fumigatus*, and the inhalation of silica-containing dust could cause silicosis, another persistent and damaging lung disease. The spreading of faeces on the land increased contact with pathogens directly or indirectly through the plants they fertilised, and typhoid, roundworm, hookworm and amoebic dysentery could all be transmitted in this way (Cockburn, 1971).

2.2.3 The Urban Environment

The introduction of specialist craft, trade and religious centres, characteristic of an urban environment, once again resulted in a shift in the subsistence base, with population movement and occupational hazards adding to the problems of overcrowding and poor hygiene. Pre-industrial urban centres, such as those in the later medieval period, relied on the rural hinterland to provide the food for the increasing population of specialists inhabiting the urban areas (Storey, 1992). Metabolic diseases such as rickets and scurvy increased due to dietary deficiencies and lack of sunlight in overcrowded accommodation (Fildes, 1986b).

Exploration and expansion into previously unexplored areas introduced new diseases to unprotected hosts, and an increase in trade and migration of people from other areas provided the possibility of new diseases being introduced to the community. This constant supply of fresh hosts, in the form of newborns and immigrants, provided the ideal breeding ground for infection. Respiratory and gastrointestinal infections, cholera, typhus, typhoid, pneumonic plague, measles and smallpox flourished in the unsanitary conditions where food, milk and the water supply were liable to contamination (Mims, 1980; Woods and Woodward, 1984). Population dependent diseases such as tuberculosis began to thrive with a ready accessibility to new hosts in overcrowded living quarters. As more hosts became immune to disease, infections became endemic (McGrath, 1992). This change in the pattern of disease in the medieval period has been

illustrated in the documentary evidence and skeletal material, where diseases such as leprosy, tuberculosis and treponematosis became more prevalent (Manchester, 1992). Hudson (1965) argues that cultural and social changes in the urban environment resulted in increased sexual promiscuity and prostitution, which contributed to the development of the venereal form of treponemal disease, syphilis.

Various studies have illustrated the negative effects of overcrowding on human health and cultural stability (Ekblad, 1993; Epstein, 1982; Russell, 1970). Russell and Russell (1970) demonstrated how civil unrest, such as the Viking invasions and disease epidemics like the Black Death, occurred at times of greatest population pressure. Noise, overcrowding, polluted air and the production of toxic chemicals would all have had an impact on both the psychological and physiological health of the individuals living in these rapidly expanding societies (Ekblad, 1993).

2.3 The Development of Urbanisation

In sixth century England, '*wics*' were major trading places situated on river trade routes in, or near, former Roman settlements. In the eighth century, many of these former *wics* had developed into fortified towns and new towns were being established; these towns were closely packed with commercial and domestic buildings, and were producing and trading goods and crafts. By the twelfth and thirteenth centuries, hundreds of new towns had been founded, aided by increased agricultural productivity, with nearly 700 English towns housing one fifth of the population (80,000-100,000) made up of craftsmen, traders and merchants (Dyer, 1989; 1997).

Initially, townspeople were provided with a quarter or half acre of land on which to build their house and outhouse. As the population grew, urban centres developed by increasing their central density, rather than expanding outwards (Ottaway, 1992). Land was increasingly divided, backyards were encroached upon and more than one family may have occupied a house, with three to four storeys added. This type of development led to problems with waste disposal, contaminated water supplies and the threat of fire (Dyer, 1989). In the late thirteenth century, migration from rural to urban areas was common. For example, York attracted 42% of migrants from a 20 mile radius, as labour shortages, due to plague deaths, provided new opportunities for women and children to gain employment and increase the family income (*ibid.*, 1989). In England, children between the ages of seven and fourteen years migrated to the towns looking for an

apprenticeship (Pelling, 1988), and women became domestic servants for the new middle classes (Goldberg, 1986). Poorer members of society and immigrants were attracted to the suburbs, initially located outside the town's defences, where polluting industries were placed to limit potential fire hazards and air pollution. The space provided by the suburbs allowed for an agricultural function, and vineyards, orchards and hop gardens were maintained there (Keene, 1975).

In the fourteenth century, war taxes, the plague, poor harvests, natural disasters, cattle fever and famine led to a gradual decline of the population in the cities (Schofield and Vince, 1994). By the fifteenth century, some of these centres had lost their urban status and people migrated to other towns or to find work in the new clothing industry, developing in previously rural areas. Further outbreaks of the plague between the fifteenth and seventeenth centuries seemed to focus on urban areas and had a detrimental effect on their development (Bean, 1962-3; Dyer, 1978). For example in 1604, an outbreak in York resulted in the loss of 10-20% of the population, but this was quickly replaced by migrants from rural areas (Dyer, 1978). The peak of the outbreaks usually occurred during the summer months when trading was at its highest and people were travelling through the towns.

2.3.1 The Urban-rural Divide

A clear distinction between *urban* and *rural* settlements was not made until the seventeenth century; prior to this urban areas had combined government buildings with vineyards and gardens. In smaller towns, differences between urban and rural diet and culture were similar, but as the towns grew they began to depend on exports to maintain the population, and the rural areas providing for the towns also grew (Schofield and Vince, 1994). Statements about the nature of rural and urban lifestyles during these periods painted rural life as noble and natural with strong family values, compared to the degenerate life in the urban areas, full of vice, indulgence, disorder and debilitating conditions (Ericksen, 1954). However, accounts about the real standard of living and health in rural areas are lacking. Some elements of the urban and rural environment were shared; rural areas provided urban centres with food, and crop failure would affect both groups. Despite having different microclimates, both areas experienced the same climatic changes and were equally vulnerable to natural disasters, and the interaction between urban and rural populations provided the opportunity for similar diseases to become endemic.

2.3.2 THE INDUSTRIAL REVOLUTION

The Industrial Revolution in the eighteenth century was characterised by rapid technological change, the establishment of new urban centres, population growth, rural-urban migration and subsequent rural depopulation (Storey, 1992). In the 1700's, the population of Leeds increased by 197%, from 1,594 to 4,651 (Pickles, 1996). Fully industrial centres were larger and more dependent on external food supplies. The gap between the social classes became more distinct and industrial advance resulted in an increase in environmental pollution.

By the 1820 s industrialisation had reached its peak and people were complaining about the effect of the urban and industrial environment on their health and were concerned about the industrial contamination of food and the water supply (Luckin, 1980). The age of onset of menstruation increased, infant mortality was high and adult mortality rates increased. Thompson (1984) paints a very graphic picture of industrial Bradford where beer shops and brothels were found on every street, adults were exposed to abject poverty and were susceptible to tuberculosis, alcoholism and venereal disease. The proximity of rural settlements to rapidly urbanising centres and the development of rural industry also had an effect on health and infant mortality. Rural areas became overcrowded as a result of immigration and the inhabitants were susceptible to the spread of disease from neighbouring cities (King, 1997).

2.3.3 MIGRATION

'As for unhealthiness, it may be supposed, that although seasoned Bodies may, and do live as long in London as elsewhere, yet new-comers and Children do not: for the Smoaks, Stinks and close Air, are less healthful than that of the country...'

(Graunt, 1662: *63*, cited in Wear, 1992: *130*)

It has been argued that due to the high levels of mortality in the urban environment, population growth in the towns could only be maintained by rural immigration (McNeill, 1979). For example, McNeill estimated that in the eighteenth century, the city of London needed an annual average immigration of 5000 people to maintain its population levels. This was seen to have led to population decline in the rural communities and to migration to the peripheries of towns, where new land could be cultivated and new diseases were encountered (*ibid.*, 1979). However, Sharlin (1978) argues that, rather than stabilising population growth in towns, rural migration added to

the number of deaths within the city by providing a vulnerable, low status population. The migrant population would have been exposed to a new variety of urban pathogens to which they had little or no resistance, and as wage-earners would delay marriage and childbirth until later years, thus lowering the birth rate and increasing death rates.

Modern studies have shown that first generation migrants have problems coping with their new environment (Kaplan, 1988). Pearson and colleagues (1993) found an increase in the level of chronic disease, alcoholism and suicide in Pacific Islanders who migrated to an urban environment. Entrance into a new environment carries high risks of disease and death for the migrants, due to their lack of adaptation, or previous exposure, to the stimuli that make up that environment. The more rapid the change, the more stressful the event (Little and Baker, 1988).

In seventeenth century London, apprentices from the countryside were notorious for contracting smallpox as soon as they arrived in the city (Pelling, 1988). In her study of skeletons from medieval York, Grauer (1991) found a higher percentage of women in the cemetery sample possibly due to higher rates of female immigration. However, these women had a lower prevalence of chronic infection, which Grauer suggests indicated that, as part of the new migratory population, they were less able to adjust to urban life and died in the acute stages of disease. Furthermore, Crawford and Goldstein (1975) examined the mortality, morbidity and fertility levels of rural immigrants living in Polish Hill in Pittsburgh between 1900-1930 s. They showed that the level of mortality and upper respiratory tract infections was higher in the immigrant settlement as a result of poor sanitary conditions, a lack of immunity to new pathogens and industrial pollution. As time passed, the Polish community adapted to their new environment, mortality and morbidity declined, more people reached reproductive age and birth rates stabilised. Physiological and psychological stress would almost certainly have been predisposing factors to disease for rural migrants in the past. In the later medieval period communities such as Wharram Percy would also have been exposed to elements of the urban environment as they migrated to the area or travelled to York and other areas to trade.

2.4 Environmental Conditions

2.4.1 Hygiene

Much of the documentary evidence for the management of English medieval towns comes from Winchester and London, where records of public concern for the odour and putrefaction of rubbish often coincided with outbreaks of the plague and official visits. Piles of refuse, or 'muck piles', in the streets would have harboured pathogenic organisms, and disease vectors such as lice, ticks, fleas and their hosts, and the byways of towns and cities were notoriously filled with sewage (Cooper, 1913). Cattle, goats and pigs were left to roam the streets and were reported to attack and injure children (Keene, 1982). Butchers and fishmongers regularly prepared their meat in the streets and blood was documented to flow down the narrow alleys (Sabine, 1933); people also complained that it was impossible to hear church services due to the barking of scavenging dogs attracted by the butcher's waste (Barnet, 1968). However, documentary evidence from medieval London show that regulations were in place to control the disposal of waste, which permitted refuse to be dumped in the River Thames (Sabine, 1933). In 1383, law also permitted the building of public and private latrines over streams acting as open sewers, which carried filth away from the city (Sabine, 1934). However, both of these regulations would have led to contamination of the water supply. The cleaning and repair of latrines and disposal of waste was usually left to the individual and it was cheaper to dump sewage into the river than pay for it to be carried away (*ibid.*, 1934). During the medieval period, it was customary for a person not to change their under clothes (and hence harbour body lice and fleas) from Michaelmas to Lady day (29th September-25th March) (Howe, 1997). The level of personal hygiene and accumulation of domestic rubbish would have depended on individual households and cannot be accounted for but, as Erasmus, the Dutch Christian humanist suggests, medieval English floors left a lot to be desired:

> "*...the bottom layer is left undisturbed, sometimes for 20 years, harbouring expectorations, vomitings, the leakages of dogs and men, ale-droppings, scraps of fish and other abominations not fit to be mentioned.*" (Keene 1982: *27*).

Environmental evidence from archaeological sites, used to reconstruct the urban-rural environment from insect and pollen remains is problematic as confounding evidence may be carried onto a site on shoes, clothing, or in a slaughtered animal's gut (Greene, 1982; Kenward, 1982). Nevertheless, Rackham (1982) found a higher concentration of pests and edible rodent species in the urban environment compared to the rural, including black and brown rats, domesticated ferrets, housemice, hares, rabbits and hedgehogs, and the remains of scavenging

birds which suggests the exposure of rubbish. At Blackfriargate in Hull, thirteenth and fourteenth century floor levels revealed parasites in rat and mice droppings (Schofield and Vince, 1994). Documents related to the employment of rat-catchers in 1356 in Hull, reveal the extent of the problem; rats would eat stored grain, and weasels and polecats were often kept to hunt them (Armitage, 1985). The analysis of a medieval cesspit from Worcester revealed that people were infected with both *Trichuris trichiura* (whipworm) and *Ascaris lumbricoides* (roundworm) (Greig, 1981). The presence of both the human and pig species of whipworm in these cesspits suggests that humans may have also contracted the parasite from eating infected meat (Jones, 1982a). Not surprisingly, 'worms' were often cited as a cause of death in medieval children (Stone, 1977), and parasitic infestation would have led to growth retardation and nutritional deficiencies (McGarvey, 1998).

Some researchers have taken evidence of official edicts on public health as suggesting that medieval cities were not filthy, and that images of medieval squalor were the creation of Victorian propaganda, designed to take attention away from the squalid conditions of the industrial cities (Lord, 1997; Thorndike, 1928). Complaints about the stench of open sewers, city cleaning and regulations controlling the dumping of butchers offal, are seen as evidence that medieval authorities were aware of the dangers that poor sanitation and piles of refuse posed to the medieval inhabitants. Butchers and fishmongers were given their own quarters of the city, principle streets were paved and night carts removed sewage (Platt, 1975). During the plague years, dogs and cats were killed and the streets cleaned, suggesting that the link between filth and disease was understood in medieval towns (Barnet, 1968). Addyman (1989) points to the establishment of stone and brick-lined rubbish pits in York as an indication that, in the fourteenth and fifteenth centuries at least, they were regularly cleaned. The introduction of clay roof tiles is also taken as an indication that in the later medieval period, living conditions had begun to improve.

Environmental conditions in eighteenth and nineteenth century cities did not improve as populations continued to grow and industrial pollution increased. Outbreaks of epidemics such as cholera were common due to contaminated water supplies (Pelling, 1983-4) and smallpox and measles were fatal childhood illnesses. Diphtheria, scarlet fever, infantile diarrhoea and whooping cough (chin-cough) were also threats to the lives of children under five years of age (Howe, 1997). Heavy drinking in towns was thought to contribute to the weak state of the infants,

and wet-nurses reportedly fed them gin to keep them quiet (*ibid.*, 1997). Finally public measures were taken to improve conditions; these included the Lighting and Cleansing Act of 1759 and the Paving Act of 1778 (Cox, 1996). However, these measures would have taken longer to be introduced in rural areas.

2.4.2 CLIMATE AND HEALTH

Temperature, wind velocity and rainfall can have a major impact on crops and animals, and can influence economic, social and political change. Proxy measures of past climate can be reconstructed using weather diaries, documentary sources of extreme weather events, movement of glaciers, insect remains, pollen assemblages, tree rings, and varves (Ingram *et al.*, 1981). These data have been used to map both global and regional temperature trends in the past.

Between the tenth and thirteenth centuries, Northern Europe, the North Atlantic, Greenland and Iceland appeared to experience a prolonged interval of warmth known as the *Medieval Warm Epoch* (AD 1000-1200) (Hughes and Diaz, 1994). This period was characterised by warm springs and dry summers, allowing vineyards to be maintained in Britain (Goudie, 1983). By the mid-fourteenth century, cold springs and wet summers predominated, and Britain entered another climatic period known as the *Little Ice Age* (AD 1400-1700) (Brimblecombe, 1982). The Little Ice Age was characterised by extremes of weather causing an increase in storms and fluctuations in summer rainfall, resulting in severe frosts, flooding and hailstorms causing serious loss of life and damage to property (Figure 2.1) (Ingram *et al.*, 1981). Lamb (1981) estimated that between the thirteenth and seventeenth centuries the growing season was shortened and crop production was reduced. The climate encouraged mold and rust to develop on grains and legumes, resulting in destruction of crops, increased grain prices, and human and animal diseases. During periods of warmer weather, insect populations grew and destroyed crops (Pearson, 1997). However, these climatic periods were regional and may have only affected certain seasons or decades (Hughes and Diaz, 1994). The climatic changes of the Little Ice Age reached their worst in 1816, recorded as '*the year without summer*' (Flohn, 1981: *312*).

Landers and Mouzas (1988) and Galloway (1985) associated the changes in the pattern of disease in summer and winter in seventeenth and eighteenth century London with climatic changes associated with the Little Ice Age, which affected the life cycle of pathogens and left the population short of food. At Christ Church Spitalfields in London, Molleson and Cox (1993)

found an association between severe winters and rickets. Climate can also have an impact on the infectivity and transmission of disease by affecting the viability of a pathogenic organism and its vectors, and the natural barriers of the host (Howe, 1997). For example, respiratory infections have been shown to increase in conditions of low humidity, leaving the nasal mucous dry and susceptible to infection (Waddy, 1952). Summer diarrhoea in infants was common in London in the seventeenth century when:

> *'from the middle of July to the middle of September these epidemic gripes of infants are so common (being the annual heat of the season doth entirely exhaust their strength) that more infants, affected with these, do die in one month than in any three that are gentle.'*
>
> (Harris, 1689 cited in Howe, 1997: *120*).

WOFULL NEWS FROM WALES
GREAT FLOOD IN MONMOUTHSHIRE, 1607

Figure 2.1 Woodcut illustrating the storms of England 1607, resulting in damage to property and livestock and serious loss of life. From Wigley et al.,*1985*:198.

Today, urban areas are known to generate their own micro-climates or *urban heat islands* where furnaces, fires, population density, air pollution and trapped heat from high rise buildings, generate excess heat. It has been estimated that medieval cities may have experienced temperatures of up to 4°C above the surrounding area (Brimblecombe, 1982; Schofield and Vince,

1994). Closer to the ground, artificial structures create an *urban canopy* resulting in an even greater increase in temperature. Howard was one of the first to notice London's '*artificial excess of heat*' in 1833 (cited in Brimblecombe, 1982: *14*). High rainfall was problematic as stone and brick buildings and limited vegetation allowed water to run-off leaving the area susceptible to flooding. This run-off, was usually polluted with rubbish and sewage, making the water supply susceptible to contamination. In fact, summers with high rainfall have been repeatedly associated with high infant mortality, as a result of water contamination (King, 1997).

2.4.3 Air Pollution

As early as 1257, documents from the Priory of Dunstable mention the pollution of urban air, and between 1285 and 1310 air pollution levels were sufficient enough to warrant four commissions to investigate the problem (Brimblecombe, 1975). The complaints about poor air quality in cities coincided with a change in fuel. Before the thirteenth century charcoal, a relatively smokeless fuel, was in common use. However, owing to fuel pressures created by the Little Ice Age and increasing industrialisation, timber became more scarce and was soon replaced by the introduction of cheaper more efficient *sea-coal* (Brimblecombe, 1978a). Coal was initially adopted by lime-burners and blacksmiths, but by the sixteenth century was eventually accepted as a domestic fuel (Brimblecombe, 1982). The more expensive charcoal was favoured by the wealthy who despised the stench released by sea-coal and considered wood smoke to be beneficial to their health. The burning of charcoal and the more smoky coal would have played a major role in polluting both the local and general environment. Sulphur dioxide and smoke, both released during the burning of coal, are synergistic pollutants, amplifying each other's detrimental effects (*ibid.*, 1982).

In the sixteenth century, the countryside was seen as the most healthy place to live (Wear, 1992), and the image of the countryside was a clean haven with fresh air, wholesome food and exercise. Air pollution in the urban centres rose steadily until concern for public safety reached its height in 1661 when John Evelyn published '*Fumifugium*' (Brimblecombe and Ogden, 1977). Increased levels of fog were responsible for a greater prevalence of diseases such as rickets (Fildes, 1986b; Foote, 1927) and air pollution was blamed for high death rates in the city (Brimblecombe, 1977).

By the nineteenth century things had not improved, and the situation in Bradford illustrates how air pollution and climate exacerbated each other's harmful effects. Poor climatic conditions and air pollution ensured that Bradford had the greatest number of people dying from respiratory

infections in all age groups, well above the national average (Thompson, 1984). The cold, rain and soot particles meant that windows were rarely opened and ventilation was poor. Babies were kept in bed rather than a separate cot to keep warm, creating opportunities for the spread of infection. In addition, the vast quantities of smoke produced by domestic and industrial burning of coal and steam power were trapped by the high hills around the valley and hung over the city (*ibid.*, 1984).

Modern studies of rural areas have shown that internal air pollution, caused by slow cooking using unprocessed biomass fuels (crop residues, dung and wood) produce levels of air pollution of the same magnitude as industrialised countries, and is associated with respiratory diseases (Albalak, 1997). Evans and Jacobs (1982) argue that air pollution can have both physiological and psychological effects on those exposed to it. High levels of pollution have been linked to respiratory diseases such as asthma, bronchitis and eye irritations, and they may lead to aggression, changes in activity patterns, decreased productivity, and memory and sensory loss. However, some research has shown that humans have the ability to eventually adapt to pollution after sustained exposure (Evans and Jacobs, 1982).

2.5 URBAN-RURAL DIET

A balanced diet is essential in order to maintain a healthy immune system and prevent disease, and the synergistic effects of nutrition and infection are well documented (Scrimshaw *et al.*, 1959). Different climates, soil types and terrain determine local diet and in the medieval period, some societies would have had access to wild foods and others to trade which would have provided a wide variety of produce throughout the year (Dyer, 1989). In her study of the Anglo-Saxon diet, Pearson (1997) argues that the consumption of wheat, rye, barley, oats and legumes provided folic acid, iron, B complex vitamins and fibre but, when compared to modern health standards, the early medieval diet was low in vitamin C. However, Dyer (1989) suggests the consumption of leeks, cabbages and apples would have prevented deficiencies severe enough to cause scurvy, that vegetables and fruit were important in the peasant diet, and that gardens were highly prized. Excessive amounts of fibre in the diet could have resulted in poor absorption of calcium, which would be especially problematic for the very young, the old and for lactating mothers (Pearson, 1997).

The consumption of meat was restricted by monastic rules, which prohibited the eating of four-legged creatures three days a week (Schofield and Vince, 1994). Herring and eel are the most common fish found in medieval and later urban deposits, and at York their consumption seemed to increase from the Anglo-Saxon to the later medieval period (Jones, 1982b). However, quantities of food consumed would have been less than today, as the animals were smaller and there were no pesticides to control crop production (*ibid.*, 1982). By the fourteenth and fifteenth centuries, there appeared to be a change in diet with an increase in wheat consumption, as people began to eat more bread rather than the traditional pottages, and ale became a popular drink (Dyer, 1989).

Studies of skeletal populations have shown an increase in dental caries dating from the sixteenth century in Britain when cane sugar (sucrose) imports increased and the first sugar factory was opened in Britian (1641). These changes meant that the once expensive commodity could be afforded by most of the population. By the end of the seventeenth century, it is estimated that the population consumed up to 20 pounds of sugar per head per year (Moore and Corbett, 1973; 1975; 1976). In addition, new milling methods led to an increase in refined flour, which contributed to the rise in dental disease. For the migratory population there may have been problems in adjusting to a new diet. Gelfand (1972) found an increase in intestinal disease in migratory populations, as a result of physiological stress and the increased consumption of refined sugar and flour.

2.5.1 The Rural Diet

Increased population density and agricultural intensification would have had a profound effect on the rural population. Manual labour meant that the nutritional requirements of the rural inhabitants were greater than those living in the urban environment. However, although estates provided rations for their tenants, the need to produce a surplus to feed the growing urban population may have meant them going without (Schofield and Vince, 1994). The introduction of the mouldboard plough and three field crop rotation system in the seventh century had meant that more land could be cultivated and a wider variety of foods grown; in addition, animals could be reared on the fallow land (Tannahill, 1973). This subsequent change in diet has been linked to increased longevity of women during the medieval period (Bullough and Campbell, 1980; Herlihy, 1975), and Bullough and Campbell (1980) argue that the new availability of meat and therefore iron, led to a decrease in deaths in childbirth.

2.5.2 The Urban Diet

In the early medieval period, developing towns had large amounts of open land within their walls. In Winchester, inhabitants grew vegetables and herbs in their gardens and kept pigs, chickens and ducks in their yards (Keene, 1983). By the sixteenth century, households were subdivided as the population increased, and gardens would have been more difficult to maintain, increasing the population's reliance on rural food production (Tannahill, 1973). In urban areas today, people are susceptible to the availability, cost and quality of the food provided by the rural hinterlands, and the cost of fuel and fresh water (Laurell *et al.*, 1977). Although medieval towns were able to store food and had more variety in their diet due to trade, including access to imported meat and fish, the poor could no longer rely on their own plots to grow food (Schofield and Vince, 1994). A study in seventeenth century Amsterdam, for example, revealed a different pattern of food debris from the rich and poor sections of the city. The rich diet appeared to consist of beef, chicken, goose, fish and oysters, whereas the poor relied on cattle and fish skulls for marrow (Izjereef, 1989).

2.5.3 Food Adulteration

Food adulteration caused considerable concern in the seventeenth century, where chemicals and metals were added to the food to add bulk and flavour. Sulphuric acid was added to vinegar, alum and crushed bone to flour, copper salts flavoured pickles and confectionery, and lead was added to wine and cider for taste, and to tea as blackening (Molleson and Cox, 1993). In 1855, public concern about food additives led to a Select Committee and the establishment of the first Food and Drug Act in 1860.

2.5.4 Infant Feeding Practices

The exposure of a newborn to its external environment is a crucial period in a human's life. The passive immunity gained from the mother is effective for only a short time if it is not replenished by the nutrients provided by the breastmilk. Colostrum, the most nutritious part of the breastmilk is present in the first two or three days after birth. It has a high content of antibodies and provides the child with maternal protection by liberating protective proteins and engulfing infective organisms in the infant's gut (Fildes, 1986a).

The transition to solid foods during weaning exposes the child to an increasing number of bacterial, viral and parasitic infections, and a diet of poor nutritional content delays growth and development. In non-industrial countries weaning diarrhoea is the primary cause of death for infants, and in Gambia it has been shown to result in growth arrest lines (Gordon *et al.*, 1963). The synergistic relationship between disease and malnutrition is well illustrated by diarrhoea diseases, which amongst other things, inhibit digestion of certain nutrients and cause anorexia. The malnourished child is at greater risk of infection and, although they grow at the same rate as their malnourished counterparts for the first six months after weaning, they eventually experience growth retardation (Beisel, 1975; Chen, 1983; Scrimshaw *et al.*, 1959).

Studies of communities making the transition to an urban environment today have shown that first generation migrants abandon breast-feeding in favour of the more convenient and fashionable formula foods. Hildes and Schaefer (1973) examined the health of Eskimos who moved from scattered hunting camps to 'urbanised' centres in Igloolik and found that the adoption of modern formulas resulted in an increase in the incidence of otitis media in the children. The abandonment of breastfeeding was a pattern also seen in Bradford Pakistani populations (Aykroyd and Hossain, 1967). Although adults maintained their own dietary habits, breastfeeding was abandoned in infants in the first few weeks as opposed to after one year in Pakistan. Amirhakimi (1974) found that, in Iran, children from high income families were less commonly breast fed, and were weaned earlier (5-6 months) compared to the urban poor and village children who were universally breast fed and weaned between 18 and 24 months. This pattern of infant feeding seems to reflect similar practices in the past.

In the medieval period colostrum was thought to be harmful due to its different colour and consistency, and was often denied the newborn child. Instead, purges of butter, oil of sweet almonds, sugar, honey, syrup or wines were fed to newborns to make them vomit and clear the mucus from their mouths and intestines. Milk-fever was a serious health risk for mothers who did not clear their breasts of milk, and infection from spoons and dishes caused potentially fatal gastrointestinal diseases, which were resolved when the child was finally put to the breast, 2-4 days after birth (Fildes, 1986a).

From the eleventh century onwards, wet-nurses became fashionable among the wealthy with children being nursed in the home or kept nearby (Fildes, 1988). However, by the seventeenth and

eighteenth centuries, wet-nursing was at its height in England and urban babies were sent as far as 40 miles away from the town to be nursed. Infants who were sent to the countryside had higher mortality rates and in the seventeenth century, the rural parish of Ware in Hertfordshire buried 1,400 children from London (*ibid.*, 1988). Children sent to the countryside would not only have been denied colostrum, but any transplacental protection (passive immunity) provided by the mother against the urban environment would have been useless in protecting against a new strain of rural pathogens. By the time the child was weaned and returned to the city, the child's antigens would be adapted for the environment in which they were weaned, and not the urban one they were returning to. For the poor, the inability to breastfeed may have led to the death of the child if a wet-nurse could not be afforded (Fildes, 1986a). However, hand feeding was also practised and children were weaned on *pap* (flour or bread crumbs cooked in milk) or *panada* (spiced bread broth). The breastmilk usually supplies zinc, an essential requirement for normal growth. However, its absorption by the body was probably hindered by the high cereal content of the pap which may have led to growth retardation (Binns, 1998). In addition, there was a high potential for contamination of the spoon and bowl used for feeding (Molleson and Cox, 1993).

In the seventeenth and eighteenth centuries, mean weaning ages dropped from 18 months to 7.25 months, a reduction that is associated with female migration to the cities to find work (Fildes, 1986a). Thompson (1984) suggests that the popularity of artificial feeding may have resulted from poor mothers in the urban environment being unable to feed their babies due to severe malnutrition. The development of a rural industry in the eighteenth century provided employment opportunities for women in textile factories with consequences on health care, breastfeeding and infant mortality. The age of weaning was brought forward or breast-feeding was interrupted, due to unpredictable income and parental illness with a subsequent increase in family size and reduced birth intervals (King, 1997).

Condensed milk was widely available in England by 1895, when consumption of artificial food doubled. However during the hot summer months, the risk of contamination once the tin was opened increased and infant diarrhoea diseases were common. Cow's milk produced in rural and urban dairies was associated with the spread of tuberculosis, scarlet fever and cholera in both urban and rural areas (Atkins, 1992; Wilson, 1986). Tough, durable feeding vessels were favoured by mothers, and bottles were often not cleaned before the next feed or between babies, providing the perfect environment for infection (Thompson, 1984). Not surprisingly, medical

writers associated weaning with disease such as rickets, gastrointestinal disease and growth retardation (Fildes, 1986a). In 1748, William Cadogan (cited in Stone, 1977) listed the major causes of infant death as fever during teething, intestinal worms (mercury and tin purges were used to treat the condition), and an inadequate milk supply from the mother or nurse. Poisoning from pewter dishes and lead nipple shields was also common.

In the nineteenth century, a change in attitudes towards breastfeeding meant that wealthy women once again fed their children, and the beneficial aspects of colostrum were recognised. Fildes (1986a) argues that this contributed to the decline in neonatal mortality in 1750.

2.6 Comparisons of Urban and Rural Health

2.6.1 Modern Studies

Issues of social status, the extent of urbanisation and the political and cultural structure of the country under analysis complicate modern studies of urban-rural health. In the USA, different socio-economic status and ethnic mixing within and between the different States make straightforward urban-rural comparisons problematic (Sobal *et al.*, 1996). There are often biases in the documentary evidence available as health surveys usually concentrate on the cities and the urban workforce (Rosen, 1988). Differences in health between urban and suburban areas also need to be investigated. In developed societies, suburban areas usually have the lowest rates of morbidity and mortality as they combine the benefits of urban life without the population pressure (Bogin, 1988b). However, in Tennessee, where greater numbers of poor Black families live in the suburbs, the opposite pattern has been reported (Woodrow *et al.*, 1978). Health differences between the rural areas and urban slums also need to be taken into account; urban slums are characterised by a low standard of hygiene, a sub-standard water supply, poor drainage and inadequate latrine and refuse collection (Banik *et al.*, 1969). In addition, modern studies have shown wealthy urban children to be better off than those in impoverished rural areas, whose health status is similar to poor children living in the urban slums (Bogin, 1988b).

Studies of urban-rural health in Britain have been inconclusive. In 1982, a study by Haynes and Bentham showed that, although there were similar patterns of disease in the urban and rural populations of southern England, the rural group had higher rates of illness. However, in northern England the picture was reversed, with the rural population displaying better health than those in

the urban centres (Phillimore and Reading, 1992). Watt and colleagues (1994) have suggested that some rural communities have limited access to medical facilities causing fewer cases of disease to be reported, and they argue that the modern assumption that rural groups have fewer social problems and are healthier is incorrect. Patients in the rural areas are less likely to consult their GP and may have different attitudes towards health and illness (Watt *et al.*, 1994).

In the USA, urban areas appear to have higher overall mortality rates, a situation which has been related to the stress of living in the urban environment and the detrimental effects of air pollution (Clifford and Brannon, 1985). However, rural areas have a higher rate of infant mortality. Defo (1996) found that in Cameroon, the differences between urban-rural infant and child mortality were largely the result of different social status. For instance, in 1987 only 12% of rural households had access to clean water as opposed to 60% in urban areas, resulting in greater infant mortality from neonatal tetanus in the rural areas. Urban mothers had greater access to medical care in the first year of their baby's life and levels between urban-rural infant mortality equalled out once hospital deliveries were taken into account (Defo, 1996). In modern day India, rural infant mortality is higher than urban mortality due to lack of access to health facilities, but in rural areas different cultural practices mean that male infants are preferentially treated and there is higher female mortality, a pattern not seen in the cities (Malhotra, 1990).

Boyden (1972) suggests that, although modern urban environments have been successful in controlling episodes of serious illness by solving their sanitation problems and maintaining good nutrition (what he calls Type A adaptation), psychological factors of well-being and self-awareness (Type B adaptation) have not been achieved. Mental illness and asymptomatic conditions such as increasingly high blood pressure suggest a level of maladaptation to the modern environment.

2.6.2 URBAN-RURAL MORTALITY

Much of the evidence for past urban-rural mortality comes from eighteenth and nineteenth century documents concerned with social reforms and combating high levels of infant deaths in the cities. Although the bills of mortality were first published in 1532 (McNeill, 1979), few studies have been carried out on earlier populations to assess differences in mortality rates between urban and rural populations, and few urban and rural cemetery populations exist for earlier periods. Under-

representation of the youngest members of these societies makes infant and child mortality rates difficult to assess. However, Storey (1988) examined the prevalence of prenatal defects and infant mortality in two samples from Mesoamerica. At Teotihuacan, a low-status densely populated urban centre, infant mortality and prenatal enamel defects were higher than at the high-status, low density site of Copan, reflecting the different stress patterns of the mothers in these two contrasting settlements. In the later periods of urbanisation at Teotihuacan (Tlajinga 33), Storey (1985) found an increase in perinatal, infant and child mortality, and attributed this to the worsening effects of urbanisation on the population.

In 1900, a discrepancy in the life expectancy of urban and rural dwellers was apparent in England. In the rural areas, the average life expectancy at birth was around 55 years of age as opposed to 45 years in the towns (Woods and Woodward, 1984). However, differences within and between urban areas existed depending on the numbers living in poverty in each part of the city (*ibid.*, 1984), and mortality rates were the highest in the factory towns of the North. Between 1850 and 1914 there was a gradual decline in mortality levels in England, despite an increase in population leading to severe overcrowding. This paradox was first explained by McKeown (1979) who argued that medical science had done little to contribute to the decline as many diseases had begun to decrease before any major advancements in chemotherapy (e.g. see Cronjé, 1984 on tuberculosis). He saw the gradual improvement of living conditions, a reduced virulence of disease and increased nutritional intake as responsible for the mortality decline. McKeown's argument has been contested by Szreter (1988; 1994) who claims that local government and the public health movement were instrumental in affecting lower mortality rates, and this issue continues to be a matter of debate (Guha, 1994; Landers, 1990; Luckin and Mooney, 1997). In truth, the decline in mortality in the cities of the eighteenth and nineteenth centuries probably resulted from a combination of improved health care services, medical advances, and public administration, which controlled the removal of sewage, provided clean water, and lit and paved the streets (Woods and Woodward, 1984). In addition to the limited opportunities for infection, it has been suggested that diseases became less virulent and epidemics less common (Ikwueke, 1984). Landers and Mouzas (1988) suggest a decline in the virulence of scarlet fever in the later eighteenth century was responsible for a change in infant death rates.

Krause (1969) argues that the decrease in mortality rates may simply be the result of a 'statistical mirage' due to the poor registration of births and deaths during the eighteenth and nineteenth

centuries. Individuals in non-Anglican burial grounds, war casualties, deaths abroad and immigrants were often omitted from the register, and deaths in hospitals, asylums and prisons were also excluded (Luckin and Mooney, 1997). In addition, the non-registration of stillbirths and non-baptised infants, even though they were included in the burial ground, affected the accuracy of the registers used by historians to estimate birth and death rates (Krause, 1969). The rise of infant mortality in 1820 may have been the result of earlier baptism that meant that more infants received funeral services and were included in the registers (*ibid.*, 1969).

2.6.2.1 Infant and Child Mortality

John Graunt (1662) was the first to extensively study levels of mortality in London using data from 1517-1519 (cited in Lancaster, 1990). He found that in non-plague years, 36% of children under six years of age could be expected to die and those urban deaths exceeded rural ones.

With a gradual decline in adult mortality rates in England in the 1880's, the high level of infant mortality became more apparent with a peak in the differences between urban and rural mortality at six months, perhaps due to the hazards of weaning (Williams and Galley, 1995). By 1907, the death rate for children between one and five years had dropped by 33%, while infant mortality remained high and averaged around 149 deaths per 1,000 live births (Dyhouse, 1978). Infant mortality was highest in the overcrowded urban and industrial mining towns of the Midlands and the North and, overall, higher in the cities than the countryside (Thompson, 1984). During this period it was widely believed that the employment of married women in towns resulted in high infant deaths (Dyhouse, 1978). In Germany, Vögele (1994) showed that larger numbers of illegitimate children were born in urban areas, and that they had less chance of survival due to poor parental care. Premature birth was also more common in the cities, perhaps due to the stresses on women employed in the tobacco industry and textiles. In a study of nineteenth century Sheffield, Williams (1992) found that children of unskilled mothers had a higher risk of death than those of professional mothers. Women who earned higher wages had better housing conditions and could protect their infants from contaminated foods during the hazardous summer months, when infant mortality rates peaked due to diarrhoeal diseases.

Williams and Galley (1995) examined infant mortality rates in urban and rural areas during the 1800 s. They found that, although there were lower levels of mortality in the rural areas, the peaks and troughs followed the same pattern in both the towns and villages (Figure 2.2). However, the

causes of infant mortality were slightly different. A report for 1891 examined differences in infant mortality between three rural counties and three industrial towns and found that the overall risk of death for infants born and reared in towns was more than double the rural picture. The majority of urban deaths were caused by diarrhoeal diseases, which killed eight times more in the urban area (39.61%) than in the rural (4.81%), and crowd diseases such as measles and scarlet fever were three times more prevalent, with tubercular diseases being double that of the rural areas. Interestingly, other respiratory diseases were twice as common in the rural than urban areas (Williams and Galley, 1995). Post-neonatal deaths from *exogenous* causes, or external hazards, were greater in urban areas, but *endogenous* (neonatal) deaths from birth defects were equal between the urban and rural communities (*ibid.*, 1995). In Germany however, neonatal mortality and still births as a result of internal health problems were higher in the rural areas (Vögele, 1994), and Malhotra (1990) states that, in the rural areas, neonatal mortality far outweighed post-neonatal mortality.

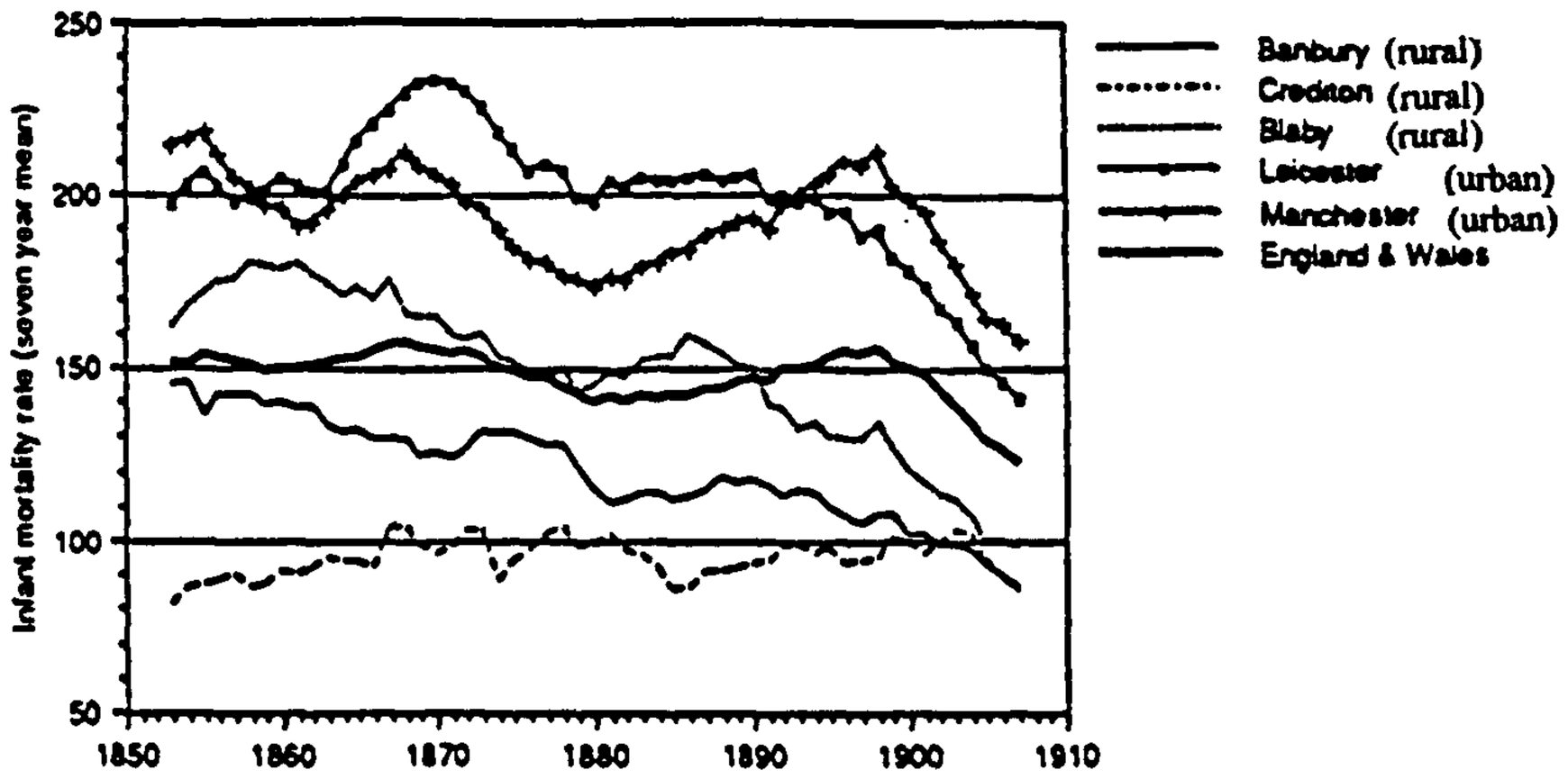


Figure 2.2. Infant mortality rates (deaths per 1,000 live births) in urban and rural areas, AD 1850-1910. From Wi and Galley, 1995: 408

Sanitation improvements in towns after the 1900 s resulted in a reversal in urban-rural infant and child mortality rates, with rural areas now showing the highest rates of mortality (Bogin, 1988a). This pattern was also reported in Germany where a peak in mortality rates in 1860-1870 was followed by a rapid decline in.the urban areas. In 1880, 91.6 per 1000 live births in rural Prussia died of diarrhoeal diseases compared to only 19.1 in the town (Vögele, 1994). In Britain, as fertility rates declined and infant mortality continued, concerns over the declining population heralded the birth of the infant welfare movement in 1906 (Dyhouse, 1978). Milk depots, free meals for nursing mothers and baby shows, with prizes for breast-fed babies, were among some of

the reforms implemented. In line with these welfare reforms, infant mortality rates dropped from 149 to 53 per 1,000 live births in 1910 (Dyhouse, 1978).

2.6.2 URBAN-RURAL GROWTH AND MATURATION

'*The* [growth] *curves of children, whether for height, weight, male or female, raised in common environments are closer, despite genetic differences, than are the curves for children raised in different environments whose genetic backgrounds are similar.'*

(Johnston *et al.*, 1985: *385*)

Guarinoni, was the first to carry out studies into urban-rural health, and in 1610 published a large volume on peasant life (cited in Tanner, 1998). He recognised that emotional stress resulted in growth retardation and reported a delayed menarche in girls from rural areas, compared to their urban counterparts. Guarinoni attributed the earlier menstruation of the city girls to their greater consumption of fatty foods and drink (Ulijaszek *et al.*, 1998).

Studies of the differences in height between children in towns and villages have been carried out since the 1870 s. During this period it was consistently found that the height of rural children surpassed that of their urban peers (Meredith, 1982). For example in 1892, Schmidt (cited in Meredith, 1982) studied the growth of German children and found that the urban group were on average, 1.8 cm shorter than their rural peers. However, by the 1950's this situation had changed, and in Meredith's (1982) survey of studies carried out between 1950 and 1980, urban children between the ages of 7-10 years exceeded the height of rural children of the same age by 2.5 cm, and in weight by 1.1 kgs. This reversal in the trend is often attributed to improvements in sanitation, nutrition and health care in the cities after the 1900's. In addition, greater energy expenditure may have contributed to the smaller heights-for-age in the rural children (Bogin, 1988a). Nevertheless, today children living in the urban slums of the developing world are still smaller in height-for-age than their rural counterparts as a result of poor nutrition and disease (Bogin, 1988b).

Studies into the variability of growth and maturation started in the 1920 s, but the use of growth as a way of understanding environmental conditions of past populations began in the 1960 s (Johnston and Zimmer, 1989). Most of these studies, carried out in North America, concentrated on Native American Indians and the impact of the transition from hunter-gathering to settled

agriculture. For example, Jantz and Owsley (1984) examined the remains of three Arikara samples from different cultural environments and found that the levels of growth corresponded to the environment from which they came, i.e. increasing morbidity and a decreasing subsistence base produced smaller growth curves. However, they did not record the prevalence of infection or malnutrition to correlate with these changing growth patterns. Changing environmental and cultural factors have been used to interpret the results of similar studies of infants and children from Indian Knoll, Illinois (Johnston, 1962), ancient Nubia (Kulubnarti and Wadi Halfa) (Hummert and Van Gerven, 1983) and the Libben Late Woodland population, Illinois (Mensforth, 1985).

Modern studies of growth in developing countries are complicated by numerous variables including different socio-economic status, genetic patterns, migration, altitude and feeding practices, that are difficult to control for and add confusion to the picture, particularly in the past, where many of these factors are impossible to account for. In 1986, Little and co-workers tried to control for socio-economic status and genetics when they examined the impact of the environment on the growth status and age at menarche in rural and urban children in Oaxaca, Mexico. They found a smaller body size and delayed age at menarche in the rural children and attributed these differences to the unpredictability of the food supply in the rural areas, as well as the dietary practices and cultural factors affecting the value placed on children. Socio-economic status also had an influence, with children from lower economic backgrounds being generally smaller and slower to mature.

In fact, most studies have shown that poverty has a greater effect on growth, regardless of whether a person came from an urban and rural environment, making any investigations into purely environmental factors very difficult (Tanner and Eveleth, 1976). For example, in nineteenth century London male children from the urban slums were up to 20 cm shorter than children from the urban upper classes (Schell, 1998). Studies of urban Nepali homeless boys, indicate that they have better growth and overall health than rural boys, and children living in urban slum areas (Panter-Brick *et al.*, 1996; Worthman and Panter-Brick, 1996). The poor growth of rural children was related to their limited nutritional intake and greater energy expenditure and, whilst the homeless boys had a degree of independence that allowed them to earn their own money and buy their own food, urban slum boys gave most of their earnings to the family (Todd *et al.*, 1996). In London during the 1700 and 1800's the Marine Society, reported retarded growth of poor

children, both before and after the growth spurt at puberty, and suggested that they were small at birth (Floud and Wachter, 1982).

In modern industrial countries, differences between growth in urban and rural areas are barely discernible (Schell, 1998) and parental education and income has a more serious effect on growth (Crooks, 1999). Tanner and Eveleth (1976) have argued that individuals living in the urban environment are genetically more heterogeneous than those in rural areas, and that this genetic interchange may affect future growth patterns. Bogin (1991) and Dettwyler (1992) have both argued that variability in growth within a population does not reflect the degree of environmental adversity, but rather the level of heterogeneity.

2.7 SUMMARY

This chapter has outlined some of the issues relating to urban and rural health, today and in the past. Differences in pathogen exposure, environmental pollution, population density and diet have been shown to result in contrasting patterns of disease and mortality. However, the migration of people from rural to urban environments, shared macroclimates and the impact of social status on infant-feeding practices and growth, make straightforward comparisons between urban and rural environments challenging today and certainly in the past, where many of these factors cannot be accounted for.

CHAPTER THREE

SKELETAL INDICATORS OF STRESS AND ADAPTATION

'...death is the ultimate indicator of failure to adapt... {it} *may be fruitfully viewed as the end result of an accumulated set of biological, behavioral and cultural challenges to the individual.'*

(Goodman and Armelagos, 1989: *231*).

3.1 CONCEPTS OF STRESS AND ADAPTATION

3.1.1 THE BIOLOGY OF STRESS

Stress is defined as any environmental stimulus that invokes a reaction in a living organism (Little, 1995). Claude Bernard (1898; cited in Little, 1995) was the first scholar to identify that living organisms had the ability to maintain constancy in their internal environment, something that was later known as '*homeostasis*'. The health of an organism depends upon this ability to regulate the internal environment and maintain homeostasis, in other words, to 'adapt' (Carruthers, 1976).

Stresses, both physical and psychological, are many and varied but the body's neuro-endocrine response to these stimuli is basically the same and involves increased adrenal-cortical activity. In response to a stimulus, the hypothalamus (at the base of the brain) stimulates the hypophysis (the small gland below it) to increase secretion of adrenocorticotrophic hormone (ACTH). This hormone stimulates the adrenal cortex and inner medulla to produce the steroids hydrocortisone, mineralocorticoids, androgens and catecholamines (adrenaline and noradrenaline), resulting in thymus atrophy, and influencing glucose and organic metabolism (Bush, 1991; Seyle, 1973).

Seyle (1950) argued that, although the body can cope with stress, the gradual build up of these responses finally takes its toll on the individual and leads to illness. Such 'stress diseases' include high blood pressure, heart disease, arthritis, asthma, ulcers and headaches (Brown, 1981). The gradual transition from resistance to stress fatigue is known as the '*general-adaptation-syndrome*', which has three stages. When first exposed to a stimulus, the individual has a low resistance and an excessive response (the alarm reaction). After repeated stress, resistance is higher and the body adapts to withstand the most common stimuli (the stage of resistance).

However, after a long period of exposure to stress, the organism can no longer adapt and is exhausted (the stage of exhaustion) (Mason, 1971; Seyle, 1973).

Baum and colleagues (1982) describe this process in terms of a pathogenic model. As an invading organism is recognised, the body responds by secreting adrenal corticosteroids, which return to normal levels if the pathogen is defeated. If the pathogen is not overcome, or recurs frequently, the body will remain in a state of constant resistance, until the pathogen has gone, or the body becomes exhausted and illness ensues. The chemical response to repeated bouts of stress, among other things, reduces the immune response and makes the individual susceptible to infection (Mazess, 1975).

3.1.2 Stress, Environment and Culture

Adaptation to environmental stress takes place on many biological levels, inducing response on the physicochemical, cellular, organ and individual level, which eventually produces changes to a population and the ecosystem (Mazess, 1975). Within a population, the ability to recover from stress is both genetically determined and culturally defined (Goodman *et al.*, 1988; Seyle, 1950; Wall, 1991). Schell (1997) argues that culture creates stresses that are not universal to all members of the society, and rather than providing a buffer, can constrain people by limiting their choices through political and economic factors. Exposure to harmful toxins, vectors of disease and overcrowding generally affect the socially disadvantaged. The very young and old members of a society are the most vulnerable, and have a limited ability to recover from a stress episode. Stinson (1985) suggests that males are less buffered against the environment than females and that in periods of stress, male foetuses have a greater mortality rate and greater growth retardation. This pattern was also seen in an earlier study of British children (Hewitt *et al.*, 1955). However, cultural practices, such as preferential infant feeding, determine whether or not an individual is exposed to, or protected from, a particular stress (Hrdy, 1990).

Stress is not entirely physiological and within each society there may be different concepts as to what is, or is not, stressful (Dressler, 1996). Culture shapes people's behaviour and, through culture, people change the environment in which they live in ways that may both advantageously and detrimentally affect their health (Brown *et al.*, 1996). For instance, urbanisation usually results in overcrowding, crowd-dependent disease transmission and environmental pollution.

3.2 STRESS IN BIOLOGICAL ANTHROPOLOGY

In 1984, Goodman and colleagues produced a stress model to illustrate the complex nature of the factors contributing to, and resulting from, physiological stress in prehistoric societies (Figure 3.1). In this model the environment acts as both a buffer and creator of stress. Culture mediates behaviour (diet, activity patterns, water use, sanitation etc.) and hence a society's ability to gain the resources necessary for survival. However, culture may also produce stress. If the individual can resist these stresses, then there is no biological response but if they succumb, physiological disruption, disease or death may ensue. This model does not take into account the psychological effects of culture and the environment, which for past societies, is more difficult to measure.

Today, stress is measured by the levels of hydrocortisone or catecholamines in urine and saliva under controlled conditions (Brown, 1981; Flinn and England, 1995). However, such direct analysis is not available for the examination of past populations and, therefore, skeletal and dental lesions thought to be indicative of physiological and psychological disruptions are used. During the stress response, neuro-endocrine activity causes the release of catabolic hormones (which release energy) so that growth, which requires energy, is inhibited (Bush, 1991). Therefore, disruption to growth and development, indicators of nutritional deficiency, and evidence of disease are recorded.

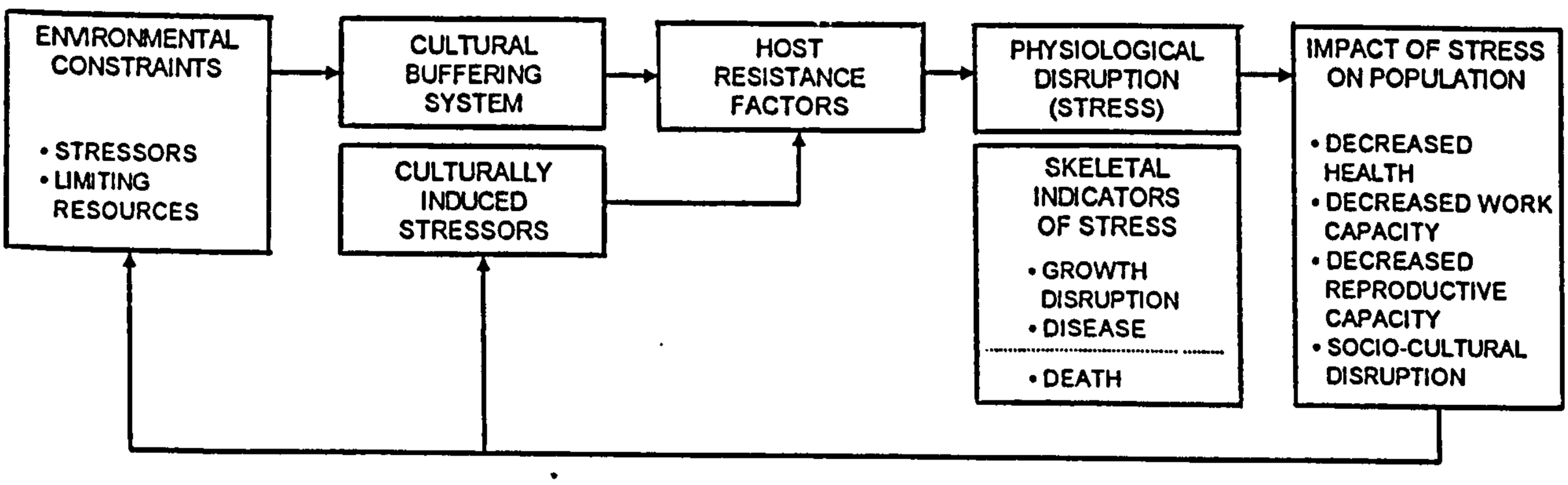


Figure 3.1. Stress model for use in skeletal populations. From Goodman et al.*, 1984b:* 14.

A commonly used indicator of stress in biological anthropology, dental enamel hypoplasias, result from a disruption in protein synthesis, caused by elevated levels of hydrocortisone, which results in a reduction in the formation of the enamel protein matrix by ameloblasts, and the hypoplastic lesion (Bush, 1991). The frequencies of these skeletal and dental lesions, within age groups and between different societies, are often analysed in an attempt to define the different cultural and environmental factors influencing health and disease. For example, Brothwell (1994) examined seven sites at York to examine the urban and rural differences in stature, craniometrics and cribra orbitalia. He calculated that at Wharram Percy (rural site) the women were 2.1 cm taller than those at St. Helen-on-the-Walls (urban) and that in the adults from Wharram Percy the prevalence of cribra orbitalia was higher (53.1%) than at both Jewbury (urban: 21.7%) and Fishergate (urban: 21.0%). However, these results were not substantiated by any statistical tests. Armelagos (1990) studied the skeletons from Dickson Mounds, Illinois during the transition from a hunter-gatherer subsistence to agricultural intensification. He found a threefold increase in iron deficiency anaemia, a threefold increase in infectious disease (periostitis) in the agricultural population, and an increase in individuals with both lesions from 6% to 40%. He also found a decrease in life expectancy for children under ten years with the lesions, and a delay in growth and development between five and fifteen years.

The aetiology of a single indicator of stress is complex, with many different conditions known to contribute to their appearance (Goodman *et al.*, 1988). However, by examining multiple indicators of stress, the focus moves away from the individual to an assessment of the population's experience of stress, mainly during childhood, when most of these indicators develop (Saunders and Hoppa, 1993; Stuart-Macadam and Kent, 1992). The age at death of those with and without stress indicators has been used to help identify those most at risk from morbidity and mortality (Cook and Buikstra, 1979; Goodman *et al.*, 1989; Grauer, 1993; Mensforth *et al.*, 1978; Van Gerven *et al.*, 1981).

3.2.1 The Osteological Paradox

In 1992, Wood and colleagues published a controversial paper that drew attention to the '*osteological paradox*.' They stated that, palaeodemographical and palaeopathological methods could only provide information on the morbidity and mortality of '*non-survivors*'.

Therefore, any attempt to reconstruct health in past populations was fundamentally flawed. They called for the use of multiple hypotheses and interpretative models in order to understand the skeletal samples under study. Their theories challenged the traditional concept that skeletons displaying skeletal lesions were the '*disadvantaged*' section of the community, and that those with no such lesions were the '*advantaged*', perhaps due to cultural and biological buffers. They created a hypothetical skeletal sample and suggested that, within this group, there were three subgroups.

The first group never experienced stress until their time of death and had no skeletal markers. The second subgroup suffered from moderate stress that left skeletal markers, but were able to recover and lived to enter the sample at a later date. The final subgroup suffered from 'severe' or acute stress that resulted in their death before skeletal markers could develop (Wood *et al.*, 1992). They stated that the subgroup that did not experience stress, and the third group exposed to acute stress, would be indistinguishable from each other in the skeletal record and, according to traditional concepts, would be wrongly identified as the advantaged section of the community. However, the second subgroup, who were strong enough to survive and recover from the condition causing the lesions, would be considered the weaker or disadvantaged group (*ibid.*, 1992).

Wood and colleagues were not the first to realise the complexity as to how and why people develop skeletal lesions. Discussing the development of palaeopathological theory in 1991, Ortner stated that the presence of skeletal lesions '*...implies a good immune response and a relatively healthy individual...*' (Ortner, 1991: *10*). Thus, in order to display pathological lesions the individual must recover from, or adapt to, the stress. The osteological paradox questioned basic and widely accepted assumptions about past health patterns, and provoked researchers to examine the methods and interpretative techniques traditionally employed in the discipline. Goodman's (1993) critical response to Wood and co-workers, highlighted the need to organise our skeletal samples into age categories and use multiple indicators of stress in order to identify any hidden

subgroups. Thus, individuals who did not show stress indicators, because they were not stressed, rather than unable to recover, would be identified by their increased longevity.

3.2.2 Non-Adults and the Study of Stress and Adaptation

The transition of a child from a stable uterine environment to the external environment, with its variety of pathogens and other stimuli, can be viewed as the first crisis in a human's life. In the womb, the foetus receives nutrients from its mother, and is protected from the external environment by the maternal immune system (Gordon, 1975; Hayward, 1978). The response of the mother to social and economic conditions indirectly influences the foetus. However, after birth, the physical and biological environment has a direct effect on the child itself. For a short time, there is a degree of continuation in the biological link between mother and child, as breast-milk supplies the child with the nutrients and passive immunity it needs to grow and to survive the new breed of pathogens it is exposed to (Hansen and Winberg, 1972).

If the child fails to adapt to its new environment, it will die. Clinically, neonatal mortality is considered to reflect the endogenous state of the infant, i.e. genetic and maternal influences, and post-neonatal mortality is seen to reflect the influence of the child's external environment (Saunders *et al.*, 1995). The ability of a given population, to provide the biocultural means for a child's survival once it is born, provides an insight into their adaptive success. Parental factors such as maternal age and nutritional status, infections, and childcare all have an effect on the child's survival (Roth, 1992) (Figure 3.2).

After the first six months of breastfeeding the breastmilk begins to loose much of its nutritional content and, if not sufficiently supplemented the child will suffer growth faltering and undernutrition, leaving susceptible to disease. However, supplementation with external foodstuffs may result in infection and potentially fatal diarrhoeal disease. This situation is known as the '*weanlings dilemma*' (King and Ulijaszek, 1999).

Goodman and Armelagos (1989) have argued that children are 'culturally unimportant', as they have no social function and can be easily replaced. However, biologically their experience of disease will have an effect on their ability to reach their full potential in adulthood, and it may eventually affect the population's ability to meet future challenges. Therefore, patterns of infant and childhood mortality and morbidity have become accepted as a sensitive barometer of

population's fitness (Mensforth *et al.*, 1978; Van Gerven and Armelagos, 1983). In biological anthropology, these ideas are widely accepted. High frequencies of stress indicators in children between the ages of 2-4 years have been related to weaning stress, and growth curves are constructed to identify stressful periods within a child's development. Nevertheless, research into comparisons of non-adults, between cultures, and in different environments through time, is limited.

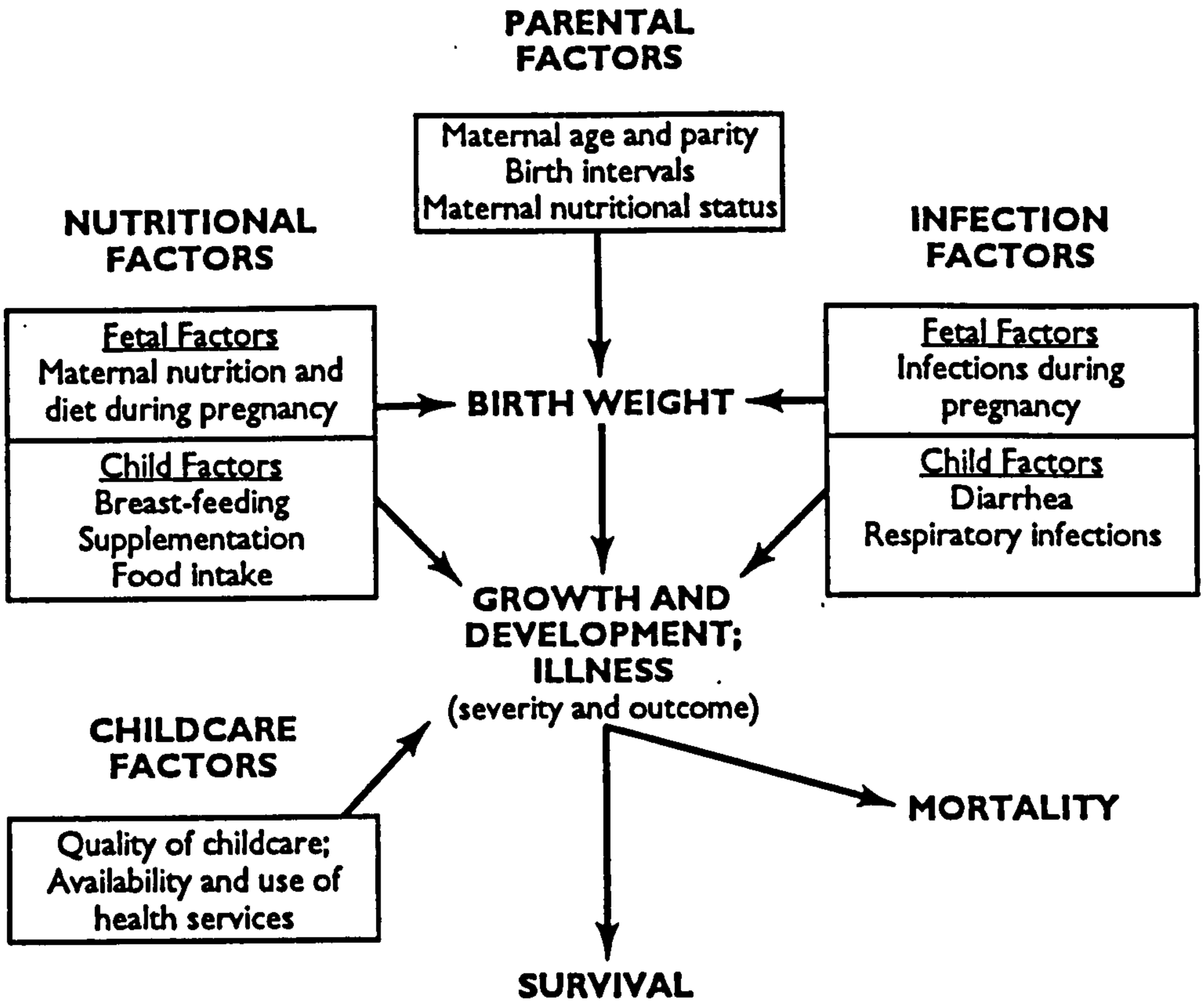


Figure 3.2 Determinants of child mortality (from Roth, 1992: 179*)*

3.3 SKELETAL INDICATORS OF NON-SPECIFIC STRESS

3.3.1 GROWTH AS AN INDICATOR OF STRESS

3.3.1.1 Introduction

Growth is a highly regulated process controlled by the endocrine system. It begins with a high growth rate after birth, which gradually slows and stabilises at three years of age. At puberty there is another period of growth acceleration which, after a period of peak velocity, slows until the epiphyseal ends of the long bones fuse and growth ceases (Karlberg, 1998). The final growth outcome is the result of a complex interaction between genetic and environmental factors. Today, growth is often used as an index of overall community health and as a guide to a population's

ability to adapt to their environment; poor growth is taken as an indicator of unfavourable conditions (Johnston and Zimmer, 1989). Secular trends showing an increase in growth between generations, are thought to indicate improvements in living conditions, enabling populations to reach greater proportions of their genetic potential (Henneberg, 1997).

Modern studies on the growth of children from different populations and social backgrounds have shown that growth is considerably adaptable and can be affected by many factors including nutrition (Metcoff, 1978), disease (Scrimshaw *et al.*, 1959), socio-economic status (Bogin, 1991), urbanisation (Tanner and Eveleth, 1976), migration (Johnston *et al.*, 1975), physical activity (Bogin, 1988a), physiological stress (Eveleth and Tanner, 1990), noise pollution (Schell, 1981), climate (Panter-Brick, 1997), and even altitude (Frisancho and Baker, 1970). A child is most vulnerable to growth retardation during the prenatal period, when maternal malnutrition (particularly protein deficiency) affects foetal size (Frisancho *et al.*, 1977), and in the postnatal period, in particular from birth to six years, when the child is experiencing major biocultural, psychological and social changes (Haas, 1990).

During a stressful period, the growth rate of a child slows until normal nourishment is resumed. The level of slowed growth relates to the severity of the episode and the age at which it occurs (Hewitt et al., 1955; Tanner, 1981). Once equilibrium has been restored, children have the ability to '*catch-up*' with their healthy peers and resume growth at an increased rate (up to three times the normal velocity) that will enable them to reach the optimum size (Tanner, 1981). This ability to increase the velocity of growth until their optimum level is reached ('*homeorhesis*') is impaired with age and, if growth is slowed for too long or too near puberty, increase growth velocity cannot fully reverse retardation (Prader *et al.*, 1963; Rallison, 1986). Although the exact mechanisms behind catch-up growth are not fully understood, it has been shown that the potential to 'catch-up' is greater if maturation is delayed and the growth period prolonged (Martorell *et al.*, 1994). However, in older individuals, accelerated growth may accelerate maturation, shortening the growth period and resulting in short stature (*ibid.*, 1994). It has been suggested that environmental stress during the childhood period may have more effect on growth than during adolescence, when genetic factors have more influence on body size (Frisancho *et al.*, 1980).

Stini (1975) referred to growth retardation as '*developmental adaptation*' and argued that this response increased an individual's chance of survival due to fewer nutritional requirements.

Seckler (1982) took this interpretation one step further and suggested that these individuals were '*small but healthy*', something that had been suggested by Frisancho and colleagues (1973) when they examined child survival rates in relation to maternal body size in Peru. They found that, although the offspring of tall women with good socio-economic conditions survived well, but in lower socio-economic groups smaller women had better success in rearing their children and, they suggest, were better adapted to their environment. However, since growth retardation has been shown to be correlated with negative health factors in both childhood and in later adulthood, short stature is considered an '*acclimatisation*' (short-term adjustment) to environmental stress factors, rather th '*adaptation*' (long-term positive adjustment)Beaton, 1989; Scrimshaw and Young, 1989).

3.3.1.2 Studies of Growth in Past Populations

When attempting to assess the health status of archaeological samples, growth data, coupled with evidence of disease and nutritional stress, can provide useful information about adaptation to certain cultural or ecological circumstances. Today, child health studies rely on anthropometric measurements such as height-for-age, weight-for-height and arm circumference (Ulijaszek *et al.*, 1998; WHO, 1986). The deviation of an individual in height-for-age, in comparison to a reference standard, is expressed as a percentage of the reference median or mean, a percentile, or as a z-score, based on the mean and standard deviation of the reference population (Haas, 1990). The reference population is derived from modern healthy children over a long period of time ('*longitudinal*' data). In archaeological samples, growth data is obtained by measuring the length of the bone shaft, or diaphysis, before the fusion of the growing ends of the bones, marking the cessation of growth. As measurements are only taken once, studies of growth in archaeological samples are based on '*cross-sectional*' data. Growth curves are constructed by estimating the age of the individual from their dental development and plotting the bone lengths against this age. The curve can then compared with modern reference curves in an attempt to measure the impact of that environment on the growth of the archaeological population.

Methods commonly used to assess the level of stress within skeletal populations include plotting skeletal age against dental age, and estimating the percentage of adult growth attainment achieved by non-adults in each age category (Humphrey, *pers. comm.*). The latter method allows for genetic differences between the populations to be normalised. Some researchers have also attempted to assess the effect of stress on maturation by comparing skeletal age with dental age (Prendergast-Moore *et al.*, 1986) as bone growth and maturation are more susceptible to

disruption by environmental factors than dental development. However, such studies rely on establishing a chronological age for the dentition, which will be based on a different standard population than the chronological age derived for bone growth. Hence, instead of comparing differences in dental and skeletal age to assess stress, they are actually comparing the differences that exist between the standard populations.

Studies examining the association between indicators of stress on the teeth and skeleton and growth retardation are relatively few, but in recent years have become more popular. In 1985, Mensforth correlated growth retardation with iron deficiency anaemia in the Libben Late Woodland sample, and attributed it to nutritional stress in the weaning period. In Britain, Mays (1985; 1995) has published several papers on the non-adults from Wharram Percy and has tested the association of growth with cortical thickness and Harris lines. Studies examining the impact of childhood stress on growth have also been carried out by Wiggins (1996) on the Barton-on-Humber non-adults, and by Ribot and Roberts (1996) who compared the prevalence of stress indicators with growth curves from a later medieval and an Anglo-Saxon population. Interestingly, few studies have shown that stress indicators are significantly correlated to growth retardation of non-adults.

3.3.1.3 Problems and Limitations

Although studies of growth in past populations are a popular way of measuring environmental stress, there are problems in attaining and interpreting a growth curve from skeletal data. Wood and colleagues (1992) do not see fluctuations in growth patterns in past populations as reflecting anything but periods of high and low mortality. They argue that when the mortality rates are high both tall and short people enter the archaeological record, providing greater mean stature for that population, but during periods of low mortality, only the frail die. As these individuals are usually shorter, they misrepresent the survivors as a short population.

In order to understand and locate periods of growth retardation for a skeletal sample, growth curves are often compared to modern growth standards, based on healthy living children. However, Saunders and Hoppa (1993) have warned that, as short stature is associated with childhood mortality in modern populations, there will be a '*mortality bias*' in the samples of children entering the skeletal record. That is, the '*non-survivors*' will be short as a result of their disease experience and cannot be compared to modern standards or be considered representative

of the living population from which they were derived. Powers (1980) tried to remedy this, at least for the eighteenth and nineteenth centuries, by publishing growth data from known aged children from archaeological assemblages; however, comparisons between different mortuary samples assume that the same level of mortality bias, in all the sites compared, is the same. Nevertheless, Lovejoy and colleagues (1990) argue that many of the children entering the archaeological record would have died from acute infections or accidents, that would not have made an impact on their growth patterns. This argument may be supported by studies that found little or no association between skeletal stress indicators and short stature.

King and Ulijaszek's (1999) model of the '*weanling's dilemma*' suggests that if growth faltering does not occur at six months then successful food supplementation must have taken place, resulting in nutritional balance without significant levels of contamination and infection. However, studies of growth on infants in archaeological assemblages are limited as long bone length is often used to estimate the age of the child in the absence of the dentition and therefore, initial growth faltering may be hidden by subsequent 'catch-up' growth once the child has recovered. If the child dies as a result of weanling diarrhoea, they would be placed in a younger age category and any growth faltering would be disguised.

The data collected for constructing growth curves from archaeological samples suffer from considerable methodological error. Non-adult remains are fragmentary and at times when the effects of stress on growth are at their greatest, few individuals are recovered. Foetal and infant remains are frequently under-represented and fewer individuals die during adolescence. Male and female growth patterns differ, especially during the onset of puberty with females usually being two years ahead of the males (Eveleth and Tanner, 1990). Without the ability to sex non-adults, it is impossible to ascertain whether any differences in growth between populations in later age groups are the effect of environmental stress, or due to difference in the numbers of males and females contributing to the mean diaphyseal length. Skeletal growth studies use dental and skeletal ages to assign a chronological age, but this method does not take into account individual variation, where a certain age category may include early and late developers (Johnston, 1968). As archaeological data is cross-sectional, evidence of growth spurts and growth velocity are unobtainable (Humphrey, 1998) and any fluctuations in age categories from small sample sizes will merely reflect this variability.

Merchant and Ubelaker (1977) showed that the analysis of growth in a single sample, using different ageing methods, produced very different curves, and therefore a lack of standardisation prevents comparisons between studies being carried out. Modern studies have shown secular trends in growth where there has been an increase in height and weight in the same populations over time (Bogin, 1988a). Although Powers (1980) reported that the individuals included in her growth study from post-medieval Britain, were up to two years behind modern children in growth and up to six months behind in their dental development, most archaeological samples are multi-period, and therefore any secular trends will be hidden by the mean measurements. In order to limit some of the errors inherent in curves derived from archaeological populations, only comparisons between mortuary samples, from similar genetic backgrounds and using the same ageing techniques, should be carried out when trying to assess environmental impact on growth. Information on environmental conditions, levels of migration and the period of cemetery use should also be obtained.

3.3.2 Cribra Orbitalia and Porotic Hyperostosis

3.3.2.1 Pathogenesis and Aetiology

Cribra orbitalia refers to porous lesions on the orbital roof (Figure 3.3), thought to be associated with similar lesions on the cranial vault (porotic hyperostosis). These lesions result from a thinning of the outer table of the skull, a widening of the inner diploic space and a '*hair-on-end*' appearance of the trabecular structure (Britton *et al.*, 1960; Ponec and Resnick, 1984; Shahidi and Diamond, 1960). During childhood, the diploic space contains red bone marrow, and an expansion of this space is thought to result from the need of the body to produce and store more red blood cells in anaemia. The anaemia can be either genetic, due to thalassaemia or sickle cell anaemia, or acquired as the result of iron deficiency in malnutrition, chronic blood loss or parasitic infestation (Holland and O'Brien, 1997; Mensforth *et al.*, 1978; Stuart-Macadam and Kent, 1992). More recently, an increased pathogen load has been implicated in the aetiology of these lesions, and it is suggested that acute and chronic infections may stimulate the immune system to withhold iron from invading micro-organisms as a defence mechanism (Weinberg, 1974; 1992).

Today, the groups most at risk from developing iron deficiency anaemia are non-adults between the ages of six months to three years, and women between the ages of 20-30; the highest frequency of the condition usually occurs before five years of age (Goodman *et al.*, 1988; Mensforth *et al.*,

1978). Iron-deficiency anaemia is still one of the most common health problems worldwide and it is estimated that 20-25% of the world's infants suffer from the condition, which affects behaviour and psychomotor development, resistance to infection and growth (Ryan, 1997).

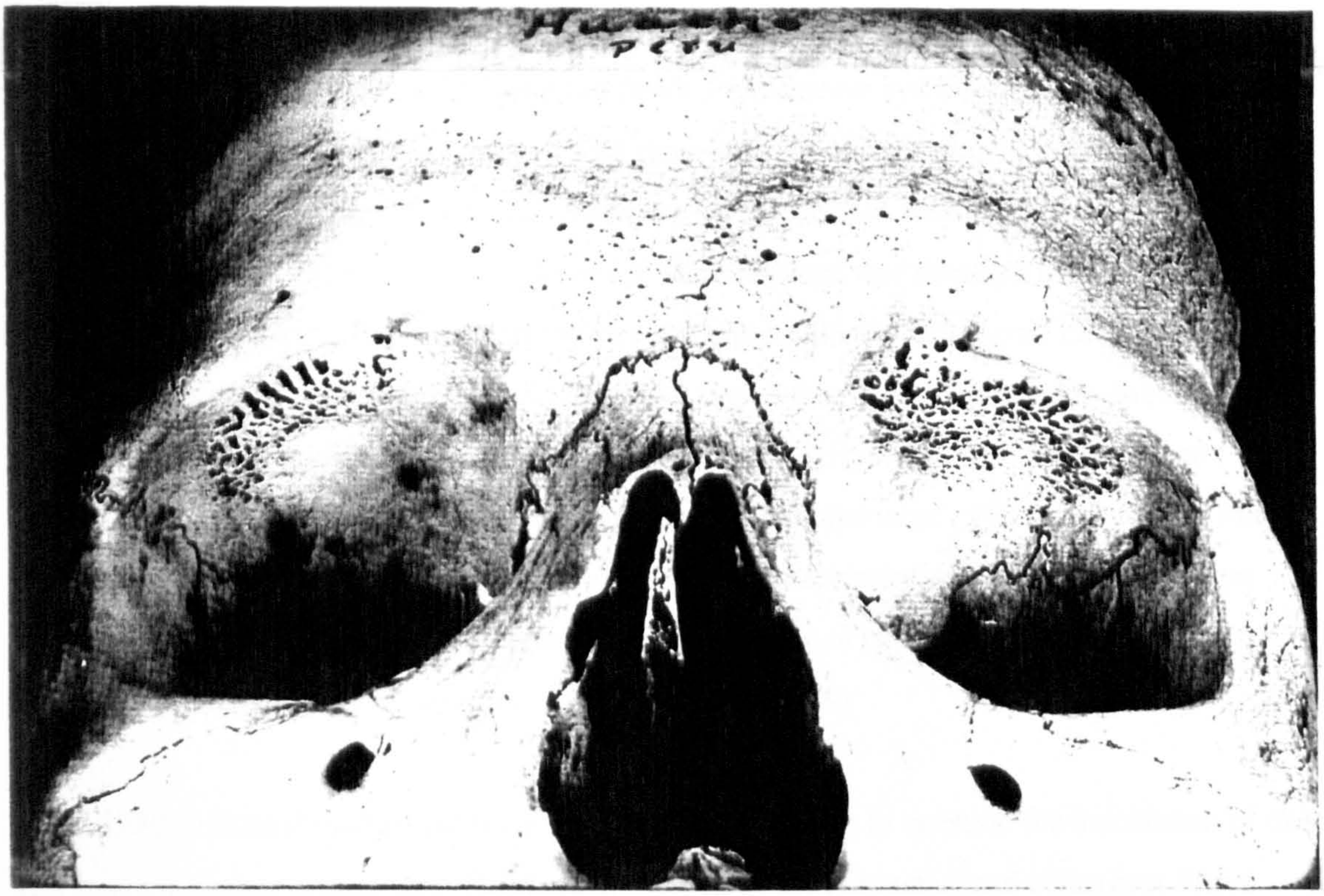


Figure 3.3 Cribra Orbitalia. From Stuart-Macadam, 1991: 108.

Anaemia in infants is not the result of low maternal iron levels during pregnancy, as this has been shown to have little effect on the iron content in breast-milk (Dallman *et al.*, 1980). Instead, anaemia is thought to be the result of a combination of reduced iron levels in maternal milk after six months, and a depletion of the iron stores held by the infant at birth (Saarinen, 1978). Premature and low birth weight babies (under 2,500g) are susceptible to iron deficiency anaemia before six months of age, as a result of lower iron stores at birth and the greater need for iron during rapid catch-up growth (Schulman, 1959).

Once breast-feeding has ceased, iron deficiency anaemia may be further exacerbated if the child is weaned on cow's milk. If this occurs before the child is six months old, intestinal bleeding can result from an irritation of the immature digestive system. After six months the body is more able to cope with the different biological properties of cow's milk, but the iron present in the milk is

less bioavailable than human milk and this eventually results in anaemia if compensatory foods are not given (Jelliffe and Blackman, 1962).

3.3.2.2 Studies in Past Populations

In the 1960 s, cribra orbitalia was thought to result from chronic eye infections. Moller-Christensen and Sandison (1963) found a high prevalence of cribra orbitalia in leprous skeletons from Denmark (63%) and intimated that the lesion was associated with eye infections as a result of the disease. Similarly in 1964, Duggan and Wells suggested that pressure from an orbital haemangioma, or lacrimal gland infections may be responsible for the lesions. Later studies of cribra orbitalia and porotic hyperostosis in skeletal populations have been used to indicate a dietary deficiency of iron, mainly as the result of a transition from a hunter-gatherer to an agricultural subsistence (Cohen and Armelagos, 1984; El-Najjar *et al.*, 1979; Gilbert and Mielke, 1985). El-Najjar (1977) believed that a dependency on maize agriculture would result in iron deficiency due to its low nutritional content and the presence of phytic acids, which inhibit iron absorption by the body (Dallman *et al.*, 1980; Saarinen, 1978).

In the 1980 s, clinical studies and trace element analyses began to question the association of diet and anaemia in past populations (Reinhard, 1992). In 1983, Fornaciari and colleagues found lower levels of iron in skeletons displaying cribra orbitalia than those without, and Glen-Haduch and co-workers (1997) associated the cribra orbitalia, not with reduced levels of iron, but with increased levels of lead which they argued, would affect homeostasis and limit the amount of iron and copper (needed to mobilise iron) stored in the body. Palkovich (1987) studied the prevalence of porotic lesions in non-adults from New Mexico and found remodelled lesions in children between 1-3 years. He argued that the maize diet resulted in malnourished mothers giving birth to low birth weight babies, who then developed anaemia before the age of six months. Walker (1986) examined the skulls of Californian Indians living off an iron rich diet of fish. Surprisingly, he found a higher prevalence of anaemia in the fisherpeople compared to those from the mainland practising subsistence agriculture, and he argued that exposure to contaminated water and fish parasites led to weaning diarrhoea and iron deficiency anaemia. Kent (1986) also suggests that the link between iron deficiency anaemia and sedentism is due to the rise in bacterial and viral infections in these environments. However, more recently, De la Rúa and colleagues (1995) returned to the question of a dietary aetiology when they found a higher percentage of cribra orbitalia in their agricultural populations than in their industrial groups. Interestingly, the link

between maize agriculture and its nutritional deficiency of iron, vitamin A and zinc has become an issue in developing countries, where today it is linked to high child mortality and poor mental and motor skills (Seymour, 1996). Holland and O'Brien (1997) argue for an approach that incorporates the effects of both diet and pathogen load when interpreting porotic hyperostosis, including cribra orbitalia, in different environments.

3.3.2.3 Association with Other Indicators of Stress

As a result of the documented synergistic relationship between malnutrition and infection, many studies have examined the relationship between cribra orbitalia and periostitis in skeletal populations. Mensforth and colleagues (1978) found an association between porotic hyperostosis and periostitis in non-adults between the ages of 6-24 months, and Palkovich (1987) found a similar correlation between periostitis, infant mortality and cribra orbitalia in New Mexico. Grauer (1993) reported that adults in Medieval York, displaying both periostitis (new bone indicative of trauma or infection) and porotic hyperostosis (including cribra orbitalia), lived 7.2 years longer than those without the lesions. She suggested that these lesions represented an individual's ability to adapt to a stress and may even strengthen them against subsequent stress episodes and lead to greater longevity. However, Van Gerven and co-workers (1981) and Mittler and Van Gerven (1994) found a high correspondence of cribra orbitalia with an increased probability of dying in infancy and early adolescence in Nubia. They estimated that those with the lesions died 15.5 years *earlier* than those without. In studies where adults are included in the analysis, the large age difference between those with and those without lesions may, in part, be due to the remodelling of lesions in the older individuals.

3.3.2.4 Problems and Limitations

One of the major problems in investigating the frequency of cribra orbitalia and porotic hyperostosis in previously recorded samples concerns the use of terminology. Some researchers do not make a distinction between the two lesions and refer to both as '*porotic hyperostosis*', a phrase first coined by Angel in 1966. This term refers to the syndrome seen on radiographs of skulls by clinicians which includes radiating trabeculae, thinning of the outer table, diploic thickening, orbital roof thickening, orbital rim changes and the underdevelopment of the frontal sinuses (Jelliffe and Blackman, 1962; Sheldon, 1936; Stuart- Macadam, 1987a). However, this term is also used to describe macroscopic changes and, in some instances, no distinction is made between purely vault or orbital lesions.

The problem arises when orbital lesions are seen in isolation, with no corresponding vault lesions. Carlson and co-workers (1974) found a prevalence of porotic hyperostosis in 0.7-4.3% of their Nubian sample compared to 21.4% displaying cribra orbitalia. Although Stuart-Macadam found an association between vault and orbital lesions at Poundbury, with 33% displaying both lesions (1987b), these lesions were severe ('hair-on-end'). Few European studies have recorded vault lesions of similar severity or frequency to the North American studies, even though orbital lesions are common. In 1991, Wiggins assessed the association of these lesions in some British skeletal populations but found no significant correlation between vault and orbital lesions. Wiggins concluded that orbital lesions may represent a milder form of iron deficiency anaemia, that the orbital and vault lesions found in the United States are of a different aetiology to those found in Europe, or that orbital and vault lesions were not of the same aetiology.

Porotic hyperostosis, involving both the vault and orbits, is commonly seen in the genetic forms of anaemia and can be distinguished from acquired iron deficiency anaemia by additional postcranial lesions (Hershkovitz *et al.*, 1997; Ortner and Putschar, 1985). Porotic hyperostosis is not pathognomonic of anaemia as it is also a feature of rickets and scurvy, but it is slightly different in expression and includes frontal bossing and the absence of the lamina dura outlining the tooth socket (Figure 3.4) (Lanzkowsky, 1968). Nevertheless, both rickets and scurvy are associated with anaemia and both types of lesions may be present.

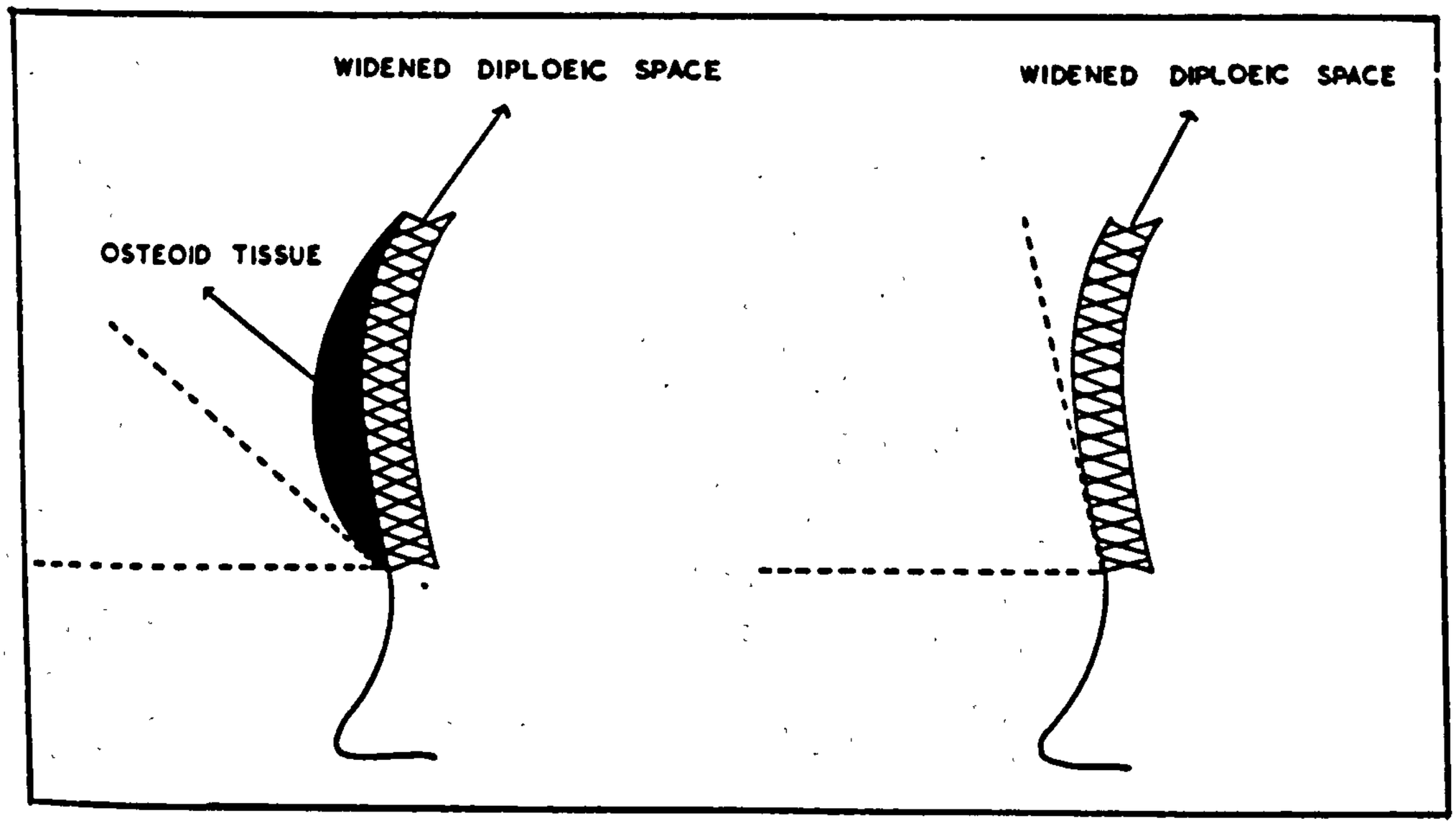


Figure 3.4 Left: lateral view of the skull in an individual with rickets and anaemia. Right: lateral view in an individual with anaemia only. From Lanzkowsky, 1968: 23.

Histological analysis and scanning electron microscopy have led some authors to conclude that conditions such as haemorrhage and inflammation may also result in similar lesions on the vault and orbital surfaces (Schultz, 1997). Cribra orbitalia and porotic hyperostosis should not be considered pathognomonic of anaemia and, at present, histological sections are needed to distinguish different causes of the lesions (Carli-Thiele, 1996). However, this type of analysis is impractical on a large scale in archaeological samples.

3.3.3 Dental Enamel Hypoplasias

3.3.3.1 Pathogenesis and Aetiology

Dental enamel hypoplasias are areas of decreased enamel thickness that occur during a disturbance of ameloblast deposition on the developing crowns of permanent and deciduous teeth (Figure 3.5). It has been proposed that elevated cortisone levels, as a result of a stress episode, inhibit protein synthesis and reduce the secretion of the enamel matrix (Rose *et al.*, 1985). The subsequent defects are seen macroscopically as either furrows or pits, and are evident microscopically as exaggerated Striae of Retzius or Wilson bands (Goodman and Rose, 1990).

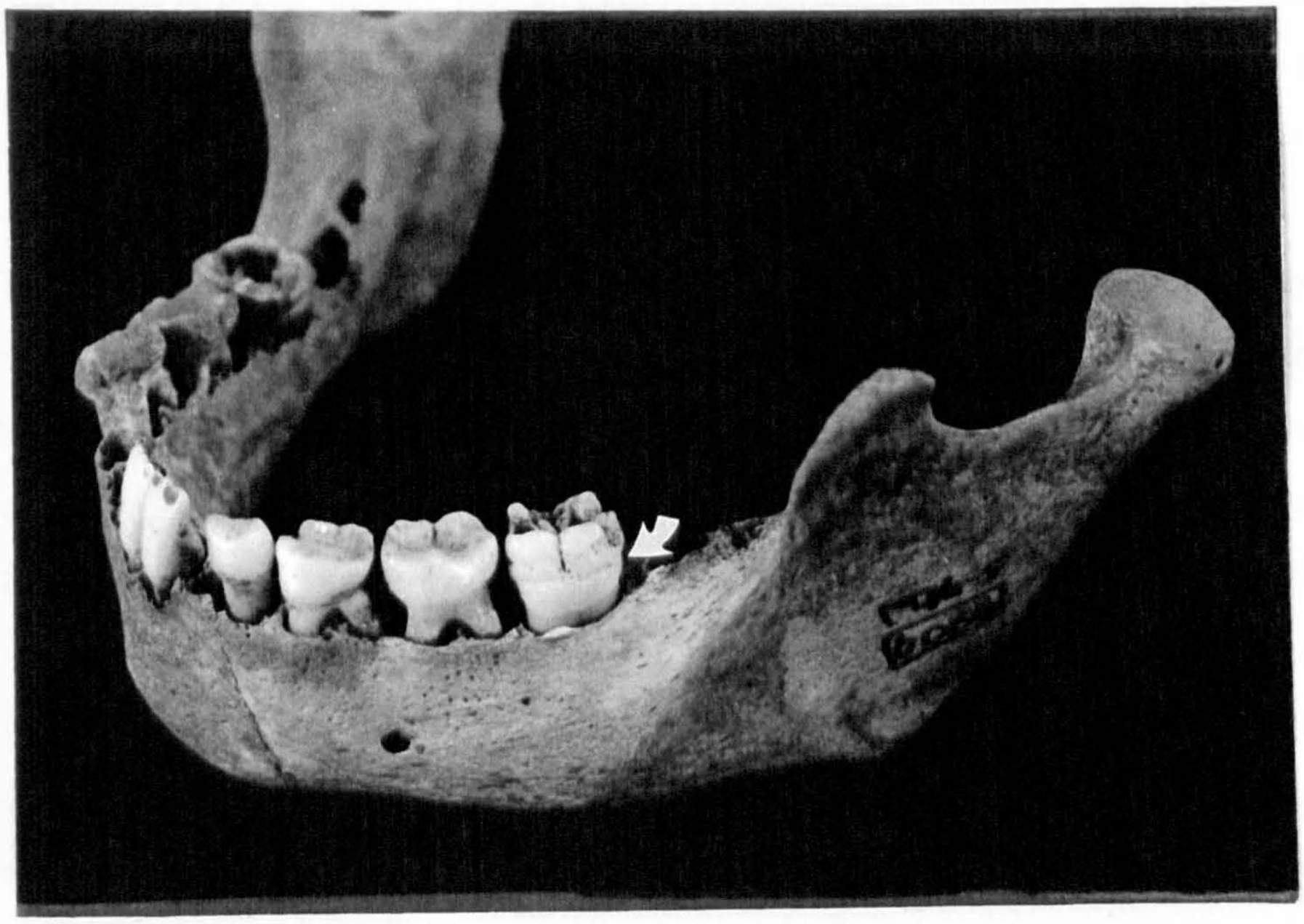


Figure 3.5 Dental enamel hypoplasias on the developing teeth of a non-adult from St. Helen-on-the-Walls, York (6056).

Dental enamel hypoplasias provide a chronological and almost permanent record of stress episodes during the pre- and postnatal period (<7 years). Sarnat and Schour (1941) concluded that, in 51% of the cases of hypoplasia in their modern sample, the exact cause of the defect could not be ascertained. However, clinical studies have linked the defects to fever, birth trauma (Kronfield and Schour, 1939), congenital syphilis (Hillson *et al.*, 1998), low birth weight (Seow, 1992), severe malnutrition (Sweeney *et al.*, 1971), rickets (Kreshover, 1960), and tuberculosis (Knick III, 1982).

3.3.3.2 Enamel Hypoplasias in Past Populations

Lanphear (1990) lists the prevalence of enamel hypoplasia in 25 different societies from hunter-gatherers to industrial populations. The highest frequencies of the defects were found in individuals from the 1800's (77-82%) and, by analysing the age of defect formation, Lanphear suggested that weaning times were earlier in the industrial populations. De la Rúa and co-workers (1995) also found an increase in enamel hypoplasias in urban and industrial societies compared to agricultural and transitional populations. However, this pattern is not universal, as Al-Abbashi (1997) examined the teeth of three groups from Jordan (the Bedouin, villagers and city-dwellers), and found the lowest frequencies of enamel hypoplaisas in the urban group. In this area, individuals living in the city were of a higher social status and were better nourished. However. Dobney (1991) did not find a consistent association between low-economic status and enamel defect formation and suggested that Asian populations were more susceptible to the defects.

The age at death of individuals with and without stress indicators has been used to help identify those most at risk from morbidity and mortality. Most of these studies have concentrated on hypoplasias in both the deciduous and permanent dentition. White (1978) noted that the age at death for African hominids with dental defects was between 4 -13 years, as opposed to 8-31 years for the non-hypoplastic individuals. However, White's sample sizes were small, with only six individuals displaying the lesion, and the differences between the mean ages were not significance tested. Since this study, a number of researchers have found a significant correlation between enamel hypoplasia formation and a lower expectancy of life (Blakey and Armelagos, 1985; Cook and Buikstra, 1979; Duray, 1996; Goodman and Armelagos, 1988; Rose *et al.*, 1978; Rudney, 1983; Stodder, 1997).

Cook and Buikstra (1979) examined the deciduous dentition of children from the Lower Illinois Valley. Their results showed that the children with defects that had developed prenatally died earlier than those who developed hypoplasias after birth, suggesting selective mortality for individuals affected in the womb. This pattern was also seen in the Dickson Mound non-adults from the USA (Blakey and Armelagos, 1985). Goodman and Armelagos (1988) also analysed data from Dickson Mounds and found an inverse association between enamel hypoplasias and mean age-at-death with those displaying multiple defects dying between 5.5-8.0 years before those without. They suggested three possible explanations for this scenario; (a) that those experiencing childhood stress had a 'weaker constitution' and as a result would have continued to experience stress into adulthood; (b) that these individuals were 'biologically damaged' by their stressful episode and would be unable to rally against any subsequent attacks that they may encounter in adulthood and, (c) that those with defects reflect a lifelong 'cultural exposure' to stress both in childhood and in later life (*ibid.*, 1988).

The above hypotheses refer to the survival of adult individuals. Thus far, little work has been carried out on the survivorship of children with enamel hypoplasias on the permanent and deciduous dentition. By concentrating on the non-adult population alone, it may be possible to more accurately assess the impact of stress on survivorship, at a time when the dental markers first appear. With the exception of Goodman and Rose (1990) and Cook and Buikstra (1979), all of these studies involved taking a mean age at death for the whole skeletal sample, which may obscure the more subtle differences in death rates within the non-adult group. Although it has been shown that stress indicators are associated with a decreased life expectancy in adulthood, this has not been tested in a non-adult sample. Those non-adults without stress indicators may be outliving the non-adults displaying them, or they may have succumbed to death before the lesions could appear.

3.3.3.3 The Weaning Hypothesis

The age of the individual at the time that a defect was formed is usually calculated by taking a measurement to the defect from the cemento-enamel junction. These measurements are then converted into a chronological age by estimating the number of years it takes the growth of the crown to be completed and into which half year of development the defect falls. Numerous studies of North American and European populations indicate the peak in development of these defects is between 2-4 years; this time period fits neatly with the age of weaning in most developing

societies. Therefore, stress caused by a change in the quality and quantity of the food supply has been implicated in the formation of these lines (Cook and Buikstra, 1979; Corruccini *et al.*, 1985; Goodman *et al.*, 1987; Lanphear, 1990; Moggi-Cecchi *et al.*, 1994; Wright, 1990). Goodman and colleagues (1984a) suggested that there was an earlier weaning age in the agricultural populations due to an earlier occurrence of hypoplasias. However, doubts about this interpretation have been raised. In 1994, Blakey and colleagues published a study carried out on the skeletons of nineteenth century African American slaves. Although it is known from documentary evidence of similar sites that the females were forced to wean their children between the ages of 9-12 months, the highest frequency of these defects occurred up to three years after that period. The study by Blakey and colleagues has raised questions about the validity of the weaning hypothesis, and proposes that other environmental factors may also be responsible.

It has been suggested that during the 2-4 year developmental period, the enamel is more susceptible to environmental disturbance. In 1984, Suckling and Thurley argued that the longer an ameloblast secretes an enamel matrix, the more vulnerable it is to insult. It may be that between the second and fourth year of enamel development the activity of the ameloblasts decreases. Goodman and Rose (1990) proposed a 'threshold model' for the development of enamel hypoplasias (Figure 3.6) and suggested that the ameloblasts were more sensitive to disruption in the middle third of the tooth.

More recently, Hillson and Bond (1997) have indicated that the position of the defect on the tooth crown may be related to the structure of the enamel layers, with earlier defects being hidden by appositional layers in the occlusal zone of the tooth. Hillson and Bond suggest that, for anterior teeth, up to 10-20% of the enamel is hidden in this way.

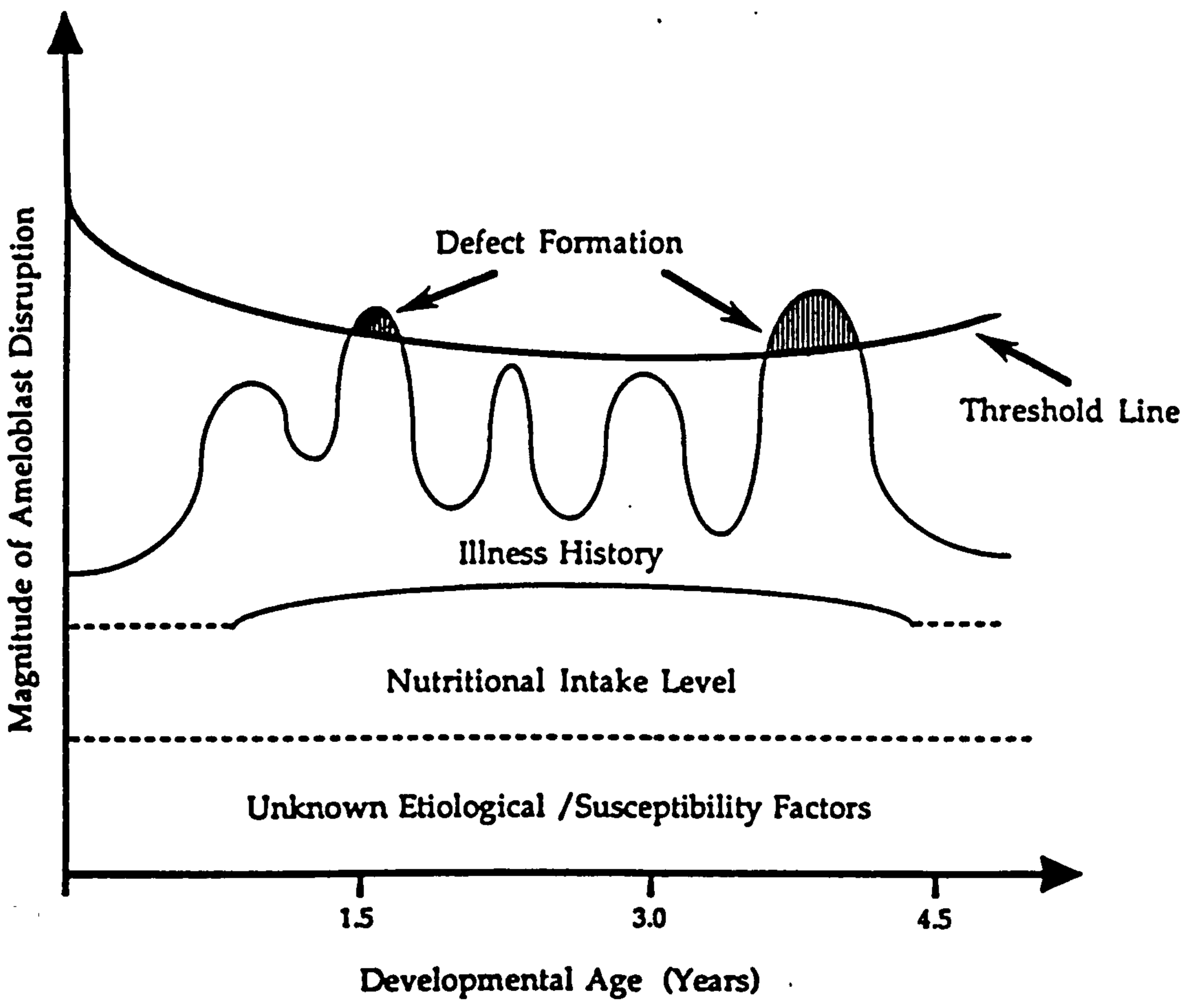


Figure 3.6 Threshold model for the formation of dental enamel defects. From Goodman and Rose, 1990: 75.

3.3.3.4 Problems and Limitations

Even if the weaning hypothesis is accepted, problems exist with the age assessment of the defects. In 1980, Goodman and colleagues constructed a table with mean ages for crown development based on data published by Swärdstedt (1966) and Massler and colleagues (1941). This method is now widely used for assessing the age at formation of a defect, but it assumes that tooth development rates in deceased ancient children are similar to today's modern healthy population. In addition, it presupposes that dental development is consistent between individuals and populations, and that tooth size variation and the type of tooth recorded has no impact on the age estimation of the hypoplasia. Comparisons between ancient and modern populations are an unavoidable problem in archaeology but can be reduced by using a standard of dental development comparable to the archaeological sample being studied. Children who entered the archaeological record, i.e. died before they reached adulthood, may be expected to have a delayed development, reflecting the impact of the environmental stresses that eventually killed them. However, the majority of children probably died as a result of acute infections or an accident,

which would not have time to affect their dental development patterns (Saunders and Hoppa, 1993). In 1990, Hodges and Wilkinson argued that tooth size variation between teeth and populations also has an impact on the age assigned to the hypoplastic defect, and Santos and Coimbra (1999) stated that there is substantial intertooth variation in the age at which the peak in defects occur.

3.3.4 HARRIS LINES

3.3.4.1 Aetiology and Pathogenesis

Transverse lines of increased radiopacity, or Harris lines, occur in the growing bones of non-adults, and have remained a popular but problematic indicator of physiological stress in ancient populations (Figure 3.7). These features were first discussed in 1903 by Ludloff (cited in Mays, 1985) who observed them in the long bones of apparently healthy individuals, but it was not until 1931 that their aetiology was fully discussed by Harris. Park described the precise mechanism behind the formation of the lines in the 1950's after carrying out experiments on rats (Park, 1954; 1964; Park and Richter, 1953).

Park (1954) found that an episode of acute or chronic stress may result in a deceleration in the development of the growth plate as the chondroblasts cease to lay down cartilage. The osteoblasts react more slowly to the stress and, for a time, continue to deposit osteoid along the static growth plate, producing a thin layer of bone along the transverse cartilage cap. This line is not visible on X-ray. When normal growth resumes, the osteoblasts recover more quickly than chondroblasts and, once again, deposit osteoid on the cartilage cap, producing a thicker lattice of dense bone, substantial enough to be visualised on a radiograph. This process continues until the maturation cycle of the chondroblasts has been completed (Acheson, 1959; Park and Richter, 1953). Hence, a line is only evident if normal growth is resumed and the individual recovers from the stress episode. In modern studies, lines usually cease to develop in non-adults after the age of 12 years in girls and 14 years in boys, possibly due to increased levels of somatotrophic hormone during bone maturation, which may compensate for any effect of disease on growth (Dreizen *et al.*, 1964). Once the epiphyses have fused, these lines can no longer develop.

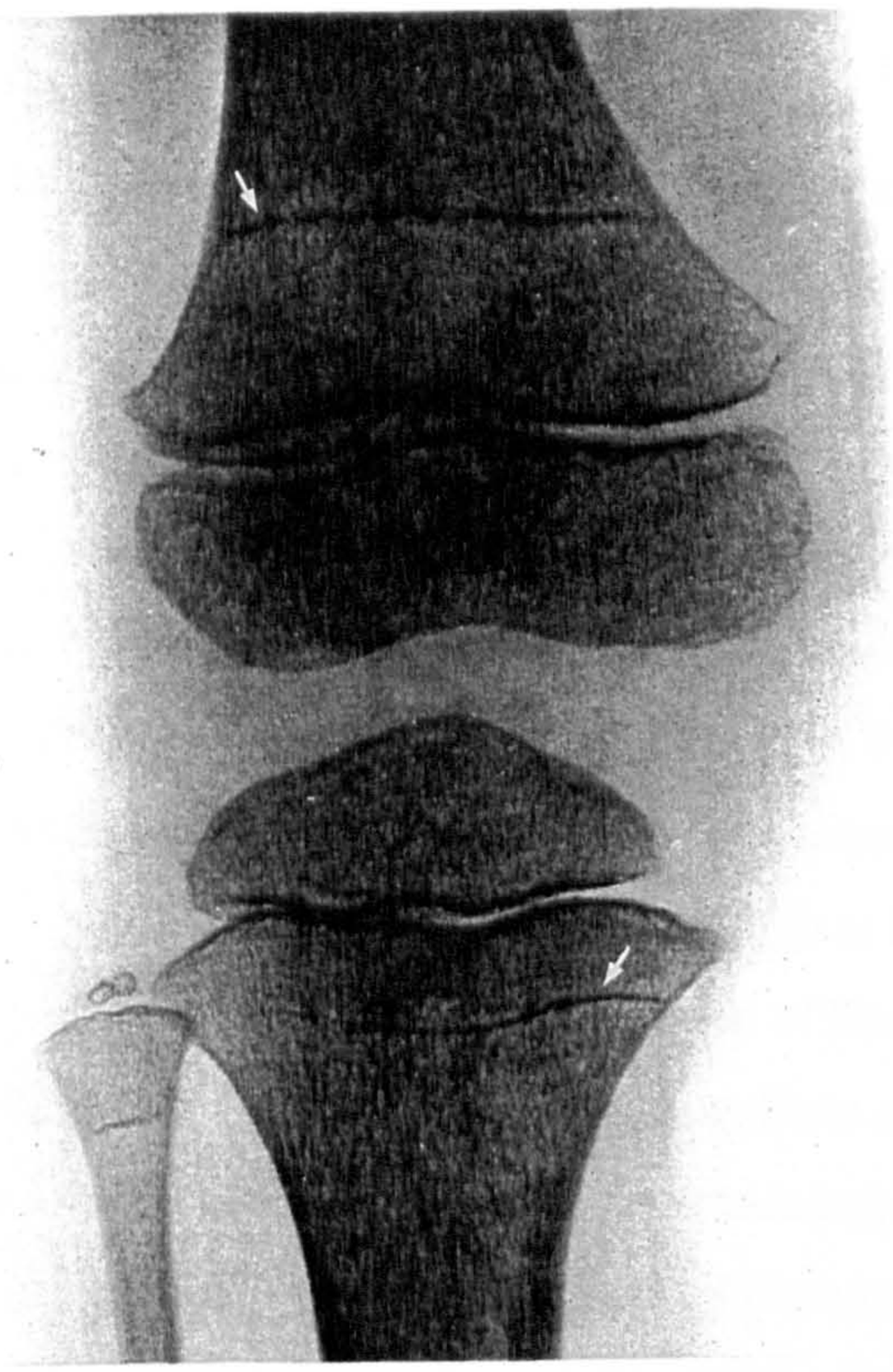


Figure 3.7 Radiograph showing a Harris line in a 6.5 years old child as the result of measles (Harris, 1933).

The exact aetiology of Harris lines has never been ascertained but starvation (Park and Richter, 1953), septicaemia, pneumonia (Acheson and Macintyre, 1958), lead poisoning, rickets, congenital syphilis and scurvy (Follis and Park, 1952) are among the many conditions associated with a slowing of growth and development of the lines. Sontag and Comstock (1938) added emotional stress to this list and Harris (1933) identified neonatal and foetal lines that he associated with birth trauma and poor maternal health. Today, the lines are often associated with weaning (Dreizen *et al.*, 1964; Goodman *et al.*, 1988) and young children are thought to be more predisposed to the development of Harris lines due to rapid growth and frequent illnesses. However, despite many experiments indicating a link between nutritional status and line development, Dreizen and colleagues (1959) showed that nutrition only had an indirect effect on the frequency of Harris line development. However, good nutrition was directly related to the child's ability to recover rapidly from the stress episode and to begin 'catch-up' growth. This process was illustrated by the greater number of fragmentary (resorbed) lines in the better nourished children.

3.3.4.2 Harris Lines in Past Populations

Harris lines have been used to establish the frequency of stress episodes in a population, either by counting the mean number of lines, the percentage of individuals affected in a population, and/or the age at which these lines occur most frequently. Several authors have developed methods to assess the age at the time of Harris line formation. One of the first attempts was by Allison and colleagues (1974); they measured the maximum length of the tibia, subtracted an arbitrary birth length (90 mm) and divided the proximal and distal ends of the bones, or the estimated growth area, into sixteen equal parts representing sixteen years of growth. However, this method was rather simplistic and assumed an equal annual growth rate and minimal male/female variability. In addition, the authors did not cite their source of long bone length at birth, or their epiphyseal fusion ages. Since then, various methods to age the formation of Harris lines have been published for the tibia (Clarke, 1982; Maat, 1984) the tibia and femur (Hunt and Hatch, 1981) and for the femur, tibia, humerus and radius (Byers, 1991). These methods employed growth standards from modern population studies in order to establish the length of the bone and, hence, the age of the individual at the time the transverse line was formed. Standards were either taken from data provided by Maresh (1955), Bock and co-workers (1973), Anderson and colleagues (1963) or Anderson and Green (1948), and methods ranged from taking two simple measurements (Byers, 1991) to using complex mathematical models.

3.3.4.3 Association with Other Indicators of Stress

Factors associated with the development of Harris lines are similar to those implicated in enamel hypoplasia formation, and for this reason attempts have been made to correlate the ages of bone and enamel line formation in skeletal populations (Clarke, 1982; Maat, 1984; Mays, 1995; McHenry and Schulz, 1976). These studies have been largely unsuccessful. In the first place, enamel does not remodel, but in an adult sample the earliest Harris lines will have been resorbed. In addition, there may be a difference in the severity of stress needed to cause a disruption in the more environmentally robust enamel. Blanco and colleagues (1974) found that children in Guatemala suffering from chronic malnutrition, displayed Harris lines and were shorter than those without such lines. They suggested that the stress that produced the Harris lines also had a detrimental effect on growth. However, studies of skeletal samples carried out to test this hypothesis have consistently shown that these lines are not associated with short stature (Carli-Thiele, 1996; Mays, 1985; Ribot and Roberts, 1996). These studies suggest that in the skeletal samples examined, children were capable of a full recovery from the stress that caused the line. Mays (1985) suggests that children displaying Harris

lines without short stature may have suffered from acute stress and were allowed enough time to 'catch-up' on the growth trajectory. In contrast, children with retarded growth and Harris lines suffered chronic stress and did not recover significantly to allow 'catch-up' growth to take place. Hence, the association of Harris lines with short stature could suggest the severity of the stress suffered.

3.3.4.4 Problems and Limitations

The use of Harris lines to estimate the frequency of stress episodes is still fraught with problems. Marshall (1968) argued that the number of lines in a child's radius did not relate to the number of insults they had suffered in the past. A year later, Gindhart (1969) showed that diseases were only followed by a line in 25% of cases and 10% of the lines occurred when no stressful episode was documented. Males and females have different rates of remodelling, and stresses as mild as an inoculation or as severe as malnutrition may cause cessation of growth and the development a line (Dreizen *et al.*, 1964). It has yet to be decided whether the thickness of a line is a measure of stress severity or longevity (Huss-Ashmore *et al.*, 1982) or whether it may be related to individual variability in the thickness of the cartilage plate or chondroblast recovery. In fact, Park argued that the position of the bone during X-ray might give a false impression of a thicker line (Park, 1954). However, it should be remembered that the individual would need to recover from the stress in order to display a line. Therefore, weaker individuals, who could not rally from stress and regain normal growth would not develop lines visible on a radiograph (Park, 1964). Some authors have suggested that line development is associated with accelerated rates of linear growth and are part of the normal growth process (Magennis, 1990; Sontag and Comstock, 1938). Nevertheless, these studies have been unable to establish whether the periods of rapid growth velocity were due to 'catch-up' growth resulting from a recovery of an insult, rather than being the causative factor. Lampl and colleagues (1992) have shown that periods of growth stasis do occur as part of the normal growth process and this may explain the appearance of Harris lines when no period of illness has been recorded.

Putting the problems with the aetiology of the lines aside, other limitations relating to the recording of these features remain. Hughes and colleagues (1996) argued that the type and side of the bone examined could affect the prevalence of the lines recorded in skeletal populations. They concluded that the distal tibia showed lines more frequently and more clearly than any other bone and that, while pairs of bones should show the presence of Harris lines, bones from the left side

had greater numbers of them. Hence, although the percentage of individuals estimated to have suffered a stress would not be affected, the frequency of the stresses would be inaccurate. In order to get the full picture of stress experience for that individual or population, radiographs of all the long bones would need to be examined (Hughes *et al.*, 1996). However, due to the poor preservation of most skeletal remains, this would prove impossible. A confounding problem was demonstrated by a study carried out by Macchiarelli and colleagues (1994) who showed that there are major discrepancies in observer error when counting the number of lines present on a bone. These worries were reiterated in 1997, when Grolleau-Raoux and colleagues found a 50% disagreement in counts for their intraobserver study and 43% for the interobserver. Until stricter rules can be applied to the identification of these lines, any attempt to compare the number of episodes of stress in different populations from previous studies would be futile. It may be better to merely score presence and absence of these lines in zones relating to different periods of development and, in order to avoid problems of line resorption, limit the study to non-adult populations.

3.4 SUMMARY

To conclude, this chapter has outlined the biological, theoretical and skeletal considerations of stress and adaptation in modern and past populations. The examination of indicators of stress in skeletal samples is an important area of research in biological anthropology. However, there is a need to reassess some of the basic assumptions about their aetiology and to be aware of the multiple factors that often contribute to their appearance. It is advisable to be conservative with the interpretations and to understand the origins of the methods used. Nevertheless, as the most vulnerable section of any community, information on the growth, mortality and morbidity of non-adults provides a unique insight in a population's ability to adapt to the environment in which they live.

CHAPTER FOUR

SKELETAL CHANGES OF INFECTION AND METABOLIC DISEASE

4.1 INTRODUCTION TO NON-ADULT PALAEOPATHOLOGY

There are many conditions commonly recorded on the adult skeleton that can be identified on non-adult remains, with the exception of neonatal material. Dental disease, specific and non-specific infections, stress indicators, trauma and metabolic diseases have all been previously recorded (Ortner and Putschar, 1985). In order for lesions produced by these conditions to be seen on the skeleton, the individual has to be compromised enough to develop the infection, but strong enough to survive the disease into its chronic stages. Acute infections, such as the plague, whooping cough, smallpox and typhus, were known to be major causes of death in the past, but killed the individual before any skeletal lesions could develop. The chronicity of a disease depends on host immunity, and the number and pathogenicity of the invading organisms.

During childhood, active growth produces a rapid turnover of bone that can both aid and hinder the diagnosis of certain conditions. For example, rickets (vitamin D deficiency) becomes apparent as large quantities of structurally inferior new bone rapidly replace the previously ossified cortex. The adult form of the disease, osteomalacia, is less evident as bone turnover occurs at a slower rate. Once the condition has resolved, accelerated growth allows the inferior bone to be quickly replaced by normal tissue causing both the macroscopic and radiographic signs of the disease to disappear from the skeleton within 3-4 months (Harris, 1931). The identification of long bone fractures in non-adult skeletons is rare compared to the rates seen in adult samples. It seems unlikely that children did not suffer injury in the past and a more likely explanation may be that they developed green-stick fractures (a partial break of the bone shaft). The highly plastic nature of non-adult bones commonly produces fractures of this type that do not result in deformation and heal quickly (Roberts and Manchester, 1995).

Both direct and indirect types of palaeopathological data can be retrieved from non-adult skeletal material. The presence of foetuses within, or expelled from, the abdominal cavity of an adult female can provide indirect evidence of obstetric health hazards, such as obstructed labour, infection or haemorrhage. The child may have been badly positioned in the womb or there may be pelvic obstruction, for example if the mother has deformities associated with vitamin D deficiency.

Congenital syphilis and tuberculosis, as well as more acute conditions, can also induce spontaneous abortion and may account for some of the foetal remains in the sample.

Mays (1993) examined the age at death of foetuses in Romano-British samples and suggested that the unusually large number of neonates, dying around the time of birth, was indicative of infanticide. This practice may be another possible reason why so few children are recovered from cemeteries during this period, and special burial areas or exposure may account for the loss of their remains. Child homicide was not confined to the Roman period, however, and documentary evidence for the burning, drowning, suffocating and poisoning of children is also available from medieval period (Damme, 1978) and the eighteenth and nineteenth centuries (Forbes, 1986; Sauer, 1978). This chapter introduces the conditions most commonly identified on the non-adult skeleton, that are indicative of chronic infection or metabolic disorders, and discusses their prevalence in the light of the social and environmental conditions that contributes to their appearance. Leprosy and treponemal disease are both specific infections visible on the non-adult skeleton. However, as no evidence for these conditions was found in the samples examined, it was decided not to discuss them in any detail.

4.2 Indicators of Non-specific Infection

4.2.1 Periosteal New Bone Formation, Osteitis and Osteomyelitis

Periosteal new bone formation (*'periostitis'*) is recognised as the deposition of a new layer of bone under an inflamed periosteum as a result of injury or non-specific infection. The periosteum is a fibrous sheath that surrounds all the bones of the skeleton, with the exception of the endocranial surface of the skull and the joints. The sheath has two layers, with the outer layer consisting of a white fibrous tissue with a few fat cells and the inner layer being made up of a dense network of fine elastic fibres (Williams and Warwick, 1980). This inner layer retains its osteoblastic capacity throughout life and is vulnerable to traumatic separation and haemorrhage. The periosteum is bound to the cortex by Sharpey's fibres, which are less numerous and shorter in children, leaving the periosteum susceptible to rupture (Caffey, 1978). In adults, the invasion of a foreign organism (commonly *Staphylococcus* and *Streptococcus*)causes inflammation, and the periosteum can become involved as a result of the direct extension of a nearby infection or, as a result of haematogenous spread of bacilli from a distant site. The inflamed periosteum

stimulates the osteoblasts to deposit osteoid on the extracortical surface of the adjacent bone (Figure 4.1).

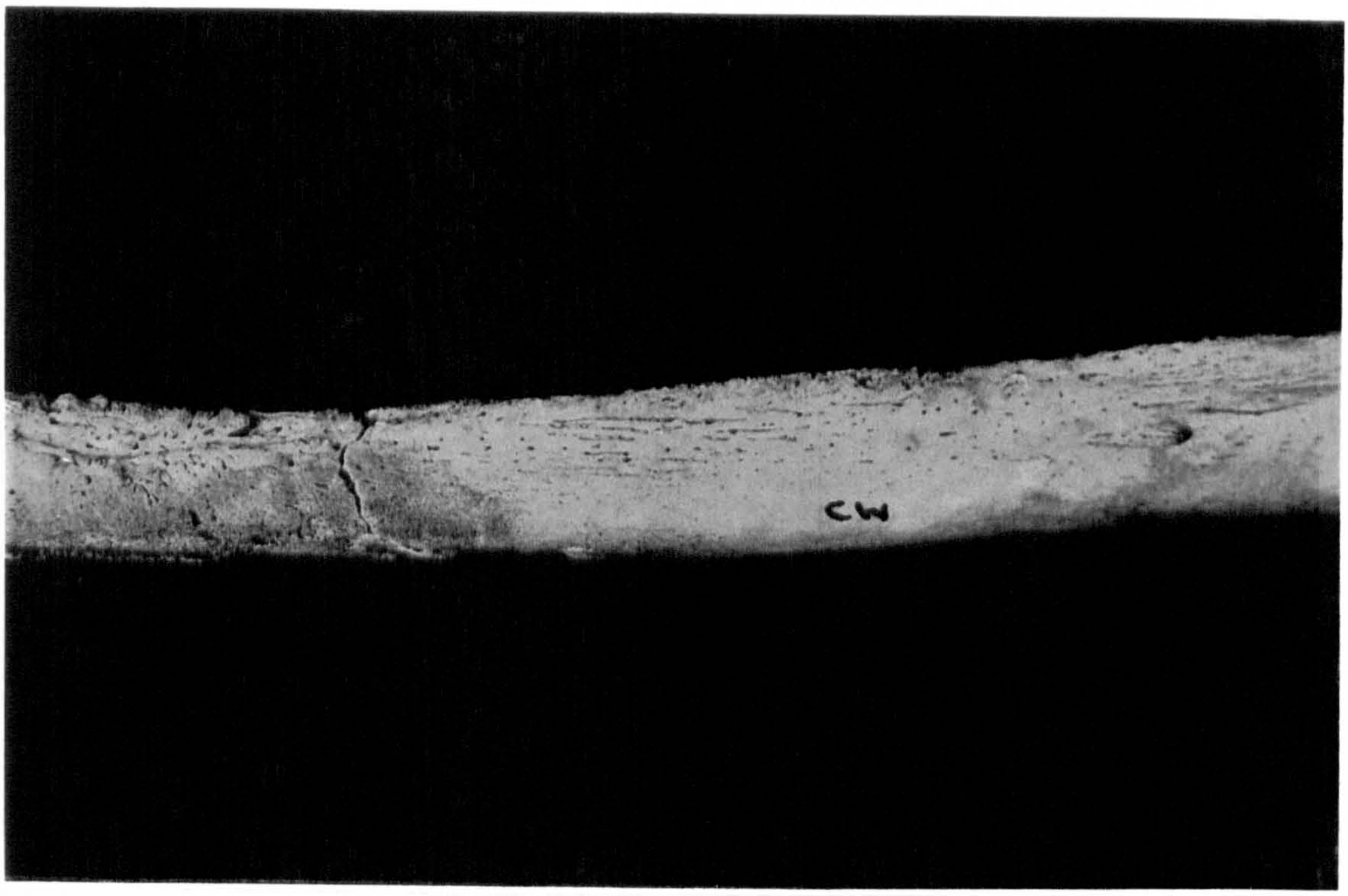


Figure 4.1 Periosteal new bone formation on the entire shaft of an adult ulna (Calvin Wells Collection, University of Bradford).

The firm attachment of the periosteum to the cortex in adults limits the spread of infection and new bone formation. However, in non-adults the periosteum may be ripped from the entire length of the shaft resulting in hypertrophy of the affected bone. Children are more susceptible to infection due to the abundant blood supply in the red bone marrow, at the ends of the long bones. A good blood supply is essential to maintain rapid growth in these areas but also transports foreign organisms to these sites, many of which rely on an abundance of iron to achieve their full growth and replication potential (Ulijaszek, 1990).

A recurring infection results in profuse sequential layers of new bone being deposited on the cortex. Initially, the bone deposited is disorganised and has a porous appearance referred to as '*woven*' bone, representing an active phase of infection. Later, the new bone layer becomes remodelled and organised with a system of Haversian canals; this smooth '*lamellar*' bone is

continuous with the original cortex and its presence is diagnostic of an infection that occurred and healed well before the person's death. A mix of woven and lamellar bone is indicative of a chronic, active infection.

The diagnosis of periostitis in non-adult skeletal remains is problematic. In the long bones, appositional (normal) growth involves the deposition of immature disorganised bone on the extracortical surface. This new bone is macroscopically identical to the woven bone deposited during an infection or after trauma. Therefore, in the youngest individuals, it is difficult to distinguish between normal and abnormal new bone and, in some cases, a diagnosis of this lesion in non-adults under the age of two years is not attempted (Ribot and Roberts, 1996). Without further investigation into the nature of the deposit and a precise guide as to where and at what age growth occurs, we may never be able to identify the true extent of inflammatory episodes in a child's life.

In children, an abundant blood supply often results in a haematogenous spread of infection, into the actual bone tissue. The shredding of the periosteum causes hypertrophy of the bone and the creation of a new sheath of bone ('*involucrum*'). Bacteria within the medullary cavity result in a necrotic focus and death of the original cortex ('*sequestrum*'), which is expelled from the bone through a draining sinus or cloaca (Figure 4.2). This type of infection is known as '*osteomyelitis*'. In more chronic cases, the infection does not produce a localised area of necrosis but spreads extensively through the bone inciting osteoblastic activity ('*sclerosing osteomyelitis*' or '*osteitis*') (Steinbach, 1966). All three types of bone infection can result from non-specific bacteria, but can also be a component of specific infections.

In the few studies that mention non-adult periostitis, it is identified as a unilateral, isolated patch of bone raised above the original cortex (Anderson and Carter, 1994; Mensforth *et al.*, 1978; Walker, 1994). However, deposits that result from a more widespread infection are rarely reported. Birth trauma, child abuse, syphilis, rickets, scurvy, hypervitamintosis A and infantile cortical hyperostosis (Caffey's disease) can all occur in the new-born and infant (Barba and Freriks, 1953; Blank, 1975; Caffey and Silverman, 1945; Malmberg, 1944-5; Snedecor *et al.*, 1935; Swerdloff *et al.*, 1970; Tufts *et al.*, 1982; Walker *et al.*, 1997), but are rarely recognised in the archaeological record. Some researchers have reported cases of osteitis and osteomyelitis in

neonates and young children from archaeological contexts, perhaps as a result of tuberculosis or congenital syphilis (Anderson and Carter, 1995; Canci *et al.*, 1991; Malgosa *et al.*, 1996).

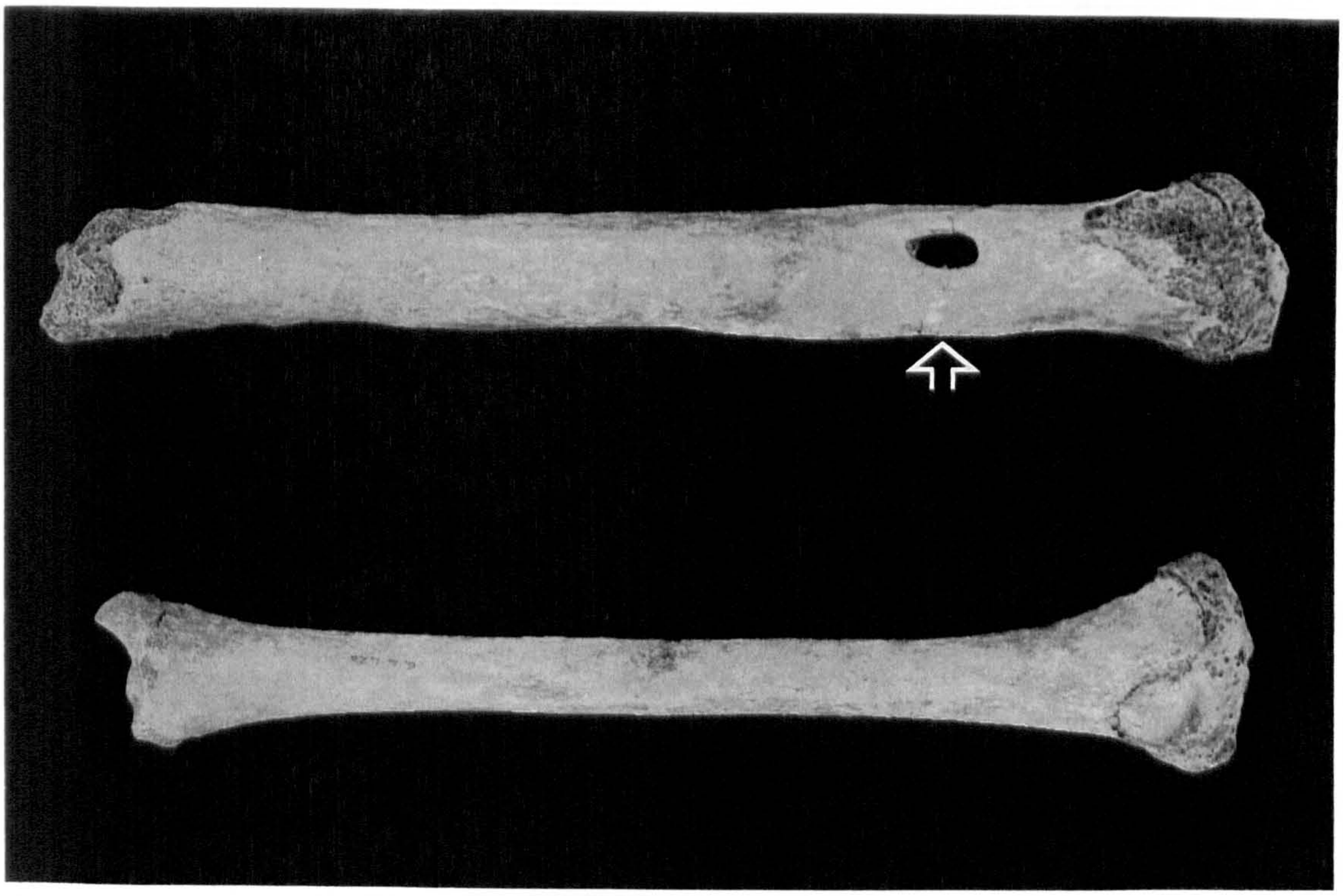


Figure 4.2. Osteomyelitis in the right tibia of a young adult with normal left side (Eccles L2a, University of Bradford).

Typhoid fever (caused by *Salmonella typhi*) results in osteomyelitis in 1% of all cases, but it leaves no other distinguishable features and cannot be diagnosed archaeologically. However, smallpox produces osteomyelitis (*osteomyelitis variolosa*) in around 20% of cases and commonly affects multiple sites including the wrists, ankles and bilateral involvement of the elbows (Jackes, 1983).

4.2.2 Chronic Maxillary Sinusitis

Maxillary sinusitis, resulting from inflammation of the mucous membrane, is a common problem in modern populations. The condition has many aetiologies including upper respiratory tract infections, poor living conditions, environmental pollution, congenital abnormalities, dental disease and, more specifically, infectious diseases including tuberculosis and leprosy (Rachelefsky *et al.*, 1984; Wald *et al.*, 1981; Wright, 1979). In children, rhinosinusitis as a result of upper respiratory tract infections is particularly prevalent. Clement and colleagues (1989) diagnosed nasal and sinus infections in 64% of children under the age of nine, and today sinus infections are

common in children attending nursery school where respiratory infections are easily transmitted (*ibid.* 1989). Horiuchi and co-workers (1981) found the condition to be more prevalent in children living in areas with high air pollution. Table 4.1 outlines the general and local factors that are associated with rhinosinusitis in children today; these conditions may also have had an influence on the condition in the past.

Maxillary sinusitis is identified in the archaeological record as deposits of new bone on the normally smooth surfaces of the maxillary antra (Boocock *et al.*, 1995). In 1995, Boocock and colleagues described, illustrated and classified the lesions found in archaeological samples (see Appendix III). However, the identification of an infection in non-adult sinuses is problematic as before the age of six the antra are not fully developed and are difficult to visualise. Until the individual is around 15 years of age, the antra continue to develop, and pits are evident on the floor of the sinus as the antra grow to accommodate the developing permanent dentition (Maresh, 1940), making lesions difficult to identify.

Few studies into maxillary sinusitis in ancient populations have been carried out, and this may be due to the fragile nature of the maxillary antra which means they often do not survive the burial environment. However in 1977, Wells carried out an examination of sinuses from the Bronze Age to the medieval period and found an increase in the condition with time. More recently, in a study of ancient urban and rural populations, environmental conditions were seen to contribute to a higher prevalence of the condition in the urban sample (Lewis *et al.*, 1995). Owing to the close proximity of the sinuses to the maxillary molars, direct infection from a dental abscess, through an oro-antral fistula, is a common aetiological factor. Therefore, the frequency of dental disease in the different populations should be taken into account when comparisons are made.

Table 4.1. Possible aetiological factors for chronic sinusitis in children (adapted from Clement et al., *1989: 527)*

GENERAL FACTORS:	
Environmental:	
	Close contact with other children*
	Climate, physical factors (temperature, humidity)
	Breastfeeding, malnutrition
Congenital and acquired abnormalities:	
	Cystic fibrosis*
	Immunodeficiency
	Immotile cilia syndrome
	Down's syndrome
	Hypothyroidism
	Congenital heart disease
	Allergy (atopy, food)
	Rhinitis
LOCAL FACTORS:	
Environmental:	
	Swimming†
	Air pollution
Congenital and acquired abnormalities:	
	Nasal malformations (choanal atresia, cleft palate)
	Nasal trauma†
	Anatomical abnormalities of the lateral wall* and septum
	Foreign bodies*
	Adenoid hypertrophy
	Dental origin
Upper respiratory tract infections:	
	Viral*
	Bacterial*
	Fungal

**factors with a clear influence on rhinosinusitis*
†*factors frequently involved*

4.2.3 ENDOCRANIAL LESIONS

Reactive new bone, usually located around the meningeal vessels on the endocranial surface of the skull in non-adults, is a relatively new area of investigation for childhood disease. These features either appear as layers of new bone on the original cortical surface, expanding around meningeal vessels, as '*hair-on-end*' extensions of the inner diploe, or as '*capillary-like*' impressions extending into the inner table of the cranium. The lesions are commonly found on the occipital bone, outlining the cruciate eminence, but have also been recorded on the parietal and frontal bones and appear to follow the areas of venous drainage (see Appendix V).

The various appearances of endocranial lesions may indicate different aetiologies; meningitis, epidural haematomas, birth trauma, neoplasia, scurvy, venous drainage problems and tuberculosis may all cause inflammation or haemorrhage of the meningeal vessels (Griffith, 1919; Kreutz *et al.*, 1995; Schultz, 1993). It is argued that the meningitis prevalent today would have killed an individual too rapidly for bone lesions to occur in the past (Fernando, *pers. comm.*). However, a study of 665 non-adult skulls from the Museum of Natural History in Vienna, found that in 111 cases where the child died from tuberculosis, 73% had 'corn-size' depressions on the endocranial surface, thought to be the result of calcified tubercles in tuberculous meningitis (Teschler-Nicola *et al.*, 1998; also see Schultz, 1999 and Jankauskas, 1999). This type of evidence may indicate that the virulence of the organism causing meningitis may have changed and, as meningitis is often a childhood disease, the distribution of these lesions in the different age categories is of interest. Cook and Buikstra (1979) identified endocranial lesions in 27% of their Lower Illinois Valley non-adults and found an association between these lesions, periostitis, dental defects and a lower expectation of life.

In the youngest skeletons, new bone formation in the form of grey woven bone, has also been identified and may represent the normal appearance of the rapidly growing skull in the infant (Williams and Warwick, 1980). However, intra-cranial haemorrhage can occur in preterm infants as a result of mineral deficiency during this rapid growth period, but the subsequent new bone formation would be difficult to distinguish from normal bone deposition (Seow, 1992). Histological analysis, however, has been used in an attempt to identify the aetiology of these lesions (Schultz, 1993). In this study, endocranial lesions were taken as indicators of a non-specific infection, and the morphological characteristics and their relationship to other stress indicators was explored.

4.2.4 Infantile Cortical Hyperostosis (Caffey's Disease)

Infantile cortical hyperostosis (ICH) affects the skeleton, the surrounding muscles and the fascias. The cause of the condition is unknown and the pathogenesis obscure. However, today it is believed to be a congenital infectious disease caused by an unknown virus transmitted through the placenta *in utero* (Caffey, 1978). The disease was first described in 1945 by Caffey and Silverman and occurs in both urban and rural environments, in all socio-economic groups, and all geographical areas. The average age of onset is nine weeks but the disease has been noted *in utero*, and has never been reported after the age of five months (Barba and Freriks, 1953; Caffey,

1978). The condition is accompanied by fever, the child becomes irritable and soft tissue swellings are evident, particularly around the mandible (Resnick and Niwayama, 1988).

In the early stages the periosteum becomes thickened and cellular, with the loss of the outer fibrous layer causing it to merge with the surrounding tissues. Osteoid is then deposited around the sheath and into the tissues but the medullary cavity retains its normal appearance. As the condition stabilises, the periosteum re-establishes its fibrous layer over the new layer of bone, which becomes incorporated into the original cortex. Remodelling begins from the endosteal surface causing the medullary cavity to widen and the bones to become more fragile (Caffey, 1978). Cortical hyperostosis is evident in all the tubular bones with the exception of the tarsals, carpals, phalanges and vertebrae. The mandible, clavicles and ulnae are the most frequently affected but the scapulae, ilia, frontal bone, parietals and orbits may all be affected with thickening and sclerosis. Of the tubular bones, the ulna is the most commonly affected and may display obvious changes next to a normal radius. There is pleurisy and costal thickening on the lateral margins of the ribs. All these lesions may be asymmetric and, unlike rickets and scurvy, the metaphyses and epiphyses are spared (Figure 4.3).

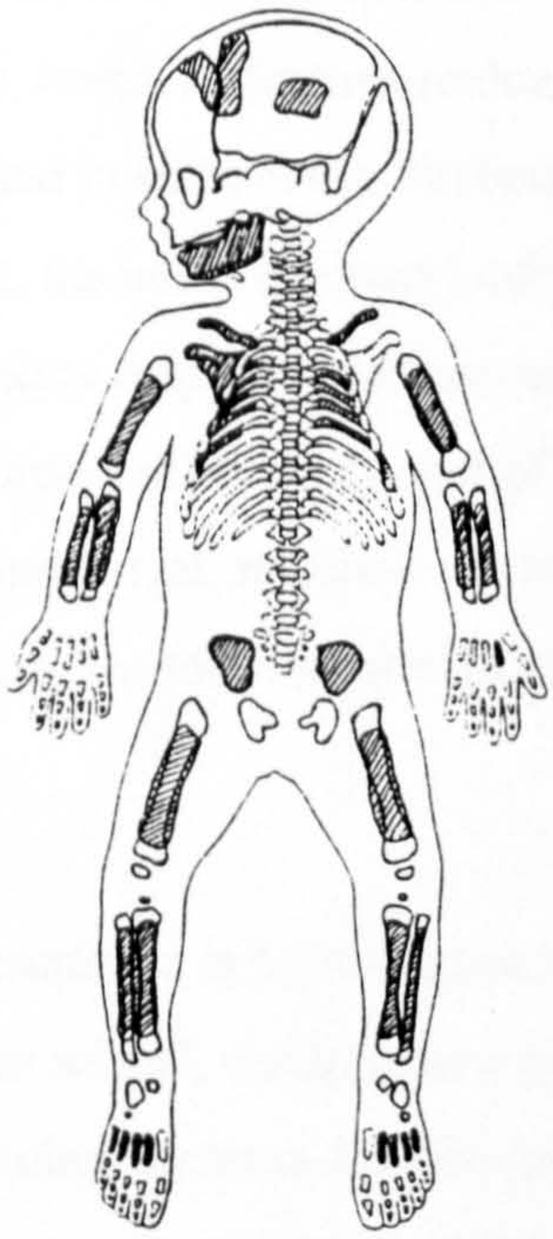


*Figure 4.3 Distribution of lesions in infantile cortical hyperostosis (after Caffey, 1978:*1434*)*

The condition can resolve within three months, but recurring forms of the disease may persist for many years and result in interosseous bridging, dislocation of the radial head, bowing of the tibiae and other severe deformities (Blank, 1975; Caffey, 1978). Cases of recurrent ICH have been reported in individuals up to 19 years of age (Swerdloff *et al.*, 1970).

Archaeological cases of ICH are extremely rare. However, in 1988 Rogers and Waldron reported two probable cases of ICH, one in a 10-18 month Romano-British infant and the other in a one year old from Anglo-Saxon England. Today, the disease is uncommon and many early signs of it could be easily missed both clinically and archaeologically. Hypervitamin osis A, trauma, vitamin deficiencies, infection and tumours also result in cortical thickening, but involvement of the mandible in these cases is unusual (Resnick and Niwayama, 1988).

4.3 TUBERCULOSIS

4.3.1 AETIOLOGY AND PATHOGENESIS

Tuberculosis (TB) is a chronic infectious disease caused by *Mycobacterium tuberculosis*. It can affect the lungs (pulmonary TB), lymph nodes(tuberculous adenitis), skin (scrofula), and intestines (gastrointestinal TB) and in some cases, the bones and joints. Tuberculosis is a childhood disease. Once infected, the initial (primary) infection may lie dormant in a healthy individual after a brief inflammatory response and become pathological later in life (secondary), when the immune status is compromised, or as a result of trauma to the affected bone or joint. However, in a more vulnerable individual, infection can lead to acute or miliary tuberculosis, which spreads throughout the body via the bloodstream, and is usually fatal (Caffey, 1978; Ortner and Putschar, 1985).

The most common mode of transmission is by inhalation of air-borne droplets containing the bacilli from an infected person or animal, resulting in a primary infection in the lungs. In 1943, 61-67% of children dying from tuberculosis in London had the pulmonary form of the disease, which commonly spread to the meninges (Sheldon, 1943). The highest incidence of the disease was in children between the age of 2-3 years, and the most susceptible had suffered from either whooping cough or measles, which were thought to lead to a reactivation of an old infection. A

primary lesion in the lungs can spread within 6-8 weeks and affect the spleen, brain, joints and kidneys. If numerous bacilli are present they can metastasise to the meninges resulting in tuberculous meningitis. The child may also developed broncho-pneumonia, and both conditions were fatal. In 1943, tuberculous meningitis was the most common form of meningitis in children and, once affected, they usually survive for up to a week before falling into coma and dying 4-5 days later (Sheldon, 1943).

In the past, it was children who were most susceptible to the gastrointestinal form of the disease (Griffith, 1919). In children, gastrointestinal tuberculosis is usually fatal in the first two or three years of life, and results from the ingestion of the human or bovine strain of the bacillus. Gastrointestinal infection, with the human strain of the bacillus, may result from an ingestion of contaminated food or, may be secondary to pulmonary tuberculosis, as infected sputum is swallowed (Sheldon, 1943). The bovine form of TB was transmitted through infected meat or milk. In 1931, even after strict rules had been applied to the storage and supply of milk, 6.7% of 'fresh' milk in England was infected with bovine TB. The bacilli cause ulceration of the intestines and may spread via the lymphatic system to the mesenteric glands and the bowel, manifesting as a generalised infection or a localised abscess. The child develops diarrhoea, suffers abdominal pain, is anaemic and becomes emaciated as a result of malnutrition (*ibid.*, 1943).

4.3.2 Skeletal Changes of Tuberculosis

Figure 4.6 illustrates the distribution of lesions on the non-adult skeleton. One of the confounding factors in the study of tuberculosis in ancient populations is that not all people dying of the disease develop skeletal lesions. Skeletal tuberculosis only occurs in around 3-5% of people with the disease (Resnick and Niwayama, 1988), but it is more common in non-adults due to the abundant blood supply of the growing bones which attracts the bacilli. Tuberculosis may affect the spine, calcaneus, hip and knee joints, and in infants affects the metacarpal and metatarsal bones ('*spina ventosa*') due to their highly vascular nature. However, these lesions are not frequently observed and may heal with little or no sign of deformity (Lincoln and Sewell, 1963; Ortner and Putschar, 1985). The lesions develop as exudate permeates the marrow spaces causing necrosis of the cancellous tissue, forming centrally localised sequestra. Periosteal reaction is limited and large involucra, common in non-specific osteomyelitis, are rare.

Lesions of the cranial vault are not common but, again, are more frequently found in children, affecting the parietal and frontal bones in the form of round foci up to 2 cm in diameter (Ortner and Putschar, 1985). Involvement of the joints may lead to destruction of the epiphysis due to abscess formation, but localised lesions may heal and small focal infections can remodel without trace. Infection of the growth plate may lead to premature fusion and deformity of the bone. Conversely, an infection in the vicinity of the growth plate may stimulate excessive growth. In the spine, an infection may begin in the centre of the vertebral body and work its way into the intervertebral disk, resulting in collapse and kyphotic deformity (*'Pott's disease'*).

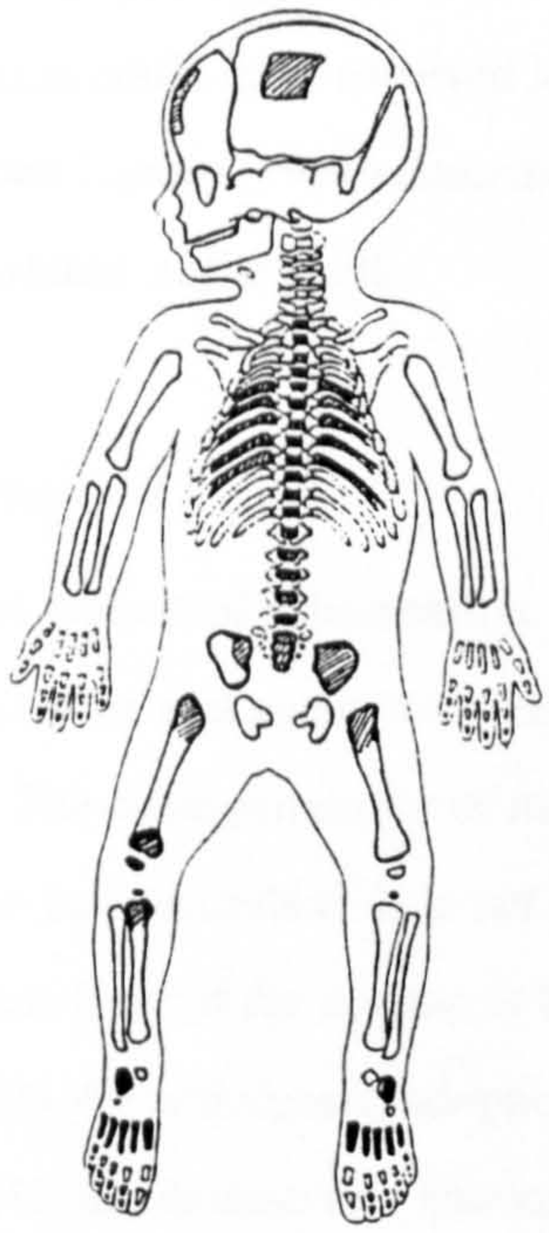


Figure 4.4. Distribution of lesions in the non-adult skeleton indicative of tuberculosis

Spinal involvement can lead to paralysis if the spinal cord is impinged upon (Lincoln and Sewell, 1963). In the pelvis, the acetabulum is more frequently affected but the ilium may become involved due to the extension of a psoas abscess causing smooth cavitation. Involvement of the shaft of long bones only occurs in childhood tuberculosis and results in massive deposits of bone due to periostitis. Congenital tuberculosis, although rare, has been documented and, by 1909 nine cases had been reported in Britain. Usually, however, the child is stillborn or dies shortly after birth (Holt, 1909).

In recent years, interest has focussed on the development of rib lesions, secondary to a lung infection, that may indicate early tuberculosis in individuals before other signs develop (Kelley and Micozzi, 1984; Roberts *et al.*, 1994, Roberts *et al.*, 1998). In addition, the possible occurrence of tuberculous meningitis for up to two weeks (without treatment) has led some authors to propose endocranial lesions as an indicator of the disease (Jankauskas and Schultz, 1995; Jankauskas, 1999; Schultz, 1999). In 1958, Lorber reported 129 cases of tuberculous meningitis, where intracranial calcifications and ossifications occurred between 18 months and three years after the onset of the disease. However, these children were receiving treatment for meningitis, which either cured them or prolonged their life long enough for lesions to develop. It is not possible to state whether children could have survived long enough in the past to develop calcifications, or if the calcifications Lorber (1958) describes at the base of the brain relate to the lesions seen on the endocranial surfaces of the skull.

4.3.3 HISTORY AND PALAEOPATHOLOGY

Tuberculosis, or consumption, is a disease of urbanisation. Poor housing and nutrition, overcrowding, low social status, climate and occupation have all been cited as causative factors (Roberts and Manchester, 1995). The close proximity of infected individuals and a low immune status is required for the disease to be transmitted. It is not known when this disease first became a parasite of humans but the human form of the disease is believed to have developed from bovine TB during the Neolithic period with the widespread adoption of agriculture. During the reign of Edward the Confessor (1042-1066), the disease was known as the King's Evil; once a year the King would hold a ceremony where he would touch the tuberculous skin lesion ('*scrofula*'), and give a sovereign to the victim. By the sixteenth century the disease was so widespread that Queen Elizabeth I stopped the ceremony claiming it was too costly (Roberts and Manchester, 1995). In the seventeenth century during non-plague years, consumption was responsible for 20% of all deaths in England and was the most common cause of death in the Western World (*ibid.*, 1995). However, signs and symptoms recognised as the King's Evil could also have been caused by syphilis, arthritis, metabolic disorders and other lung infections, and it was not until 1689 that Morton separated *scrofula* into the tuberculous and non-tuberculous forms (Chalke, 1962).

One of the major causes of tuberculosis in children between the sixteenth and nineteenth centuries is believed to be the use of cow's milk for infant feeding. During its height (1850-1860) milk was produced in urban cowsheds or on the outskirts of the town. Reports of adulterated and infected

milk were common, as the transportation of milk from the countryside could take up to 24 hours in un-refrigerated conditions before being stored, uncovered, in shops or in the home (Atkins, 1992). By the 1890's the health hazards of this method of milk production were being recognised but, despite reforms, 5.5% of milk produced in Manchester was still infected with the bacillus, and in the less strictly run rural cowsheds 17.6% of milk was affected (Cronjé, 1984). Despite the introduction of pasteurised milk, many children continued to develop tuberculosis, as it was an expensive commodity (Atkins, 1992).

By the 1870 s, improvements in sanitation in the urban centres lead to a decline in tuberculosis. However, it still remained a problem in the rural areas that did not benefit from the new reforms. Chalke (1962) argued that tuberculosis was on the increase before the advent of the industrial revolution, and had began to decline in the 1780 s before any sanitary reforms had been implemented, and at a time when cholera and typhoid were rampant. Chalke claims that the difference in the prevalence of tuberculosis in urban and rural environments was complicated by the migratory patterns of the inhabitants. It was well recognised that women were more commonly affected with the disease, and it was the women who were entering the towns to gain employment as domestic servants (Goldberg, 1986). When they became sick they would return home to the country to die. However, the common perception that the disease was contracted in the town may have been misplaced. Women leaving the countryside may have already been infected with tuberculosis, which only became manifest when they entered the stressful conditions of urban servitude. Their return home gave the impression that they had contracted the disease in the town. In fact, they may have been originally carried the disease to the cities contributing to the already high prevalence there.

4.4 Metabolic Diseases

4.4.1 Marasmus and Kwashiorkor

Although these diseases cannot be directly identified in the skeletal record, they undoubtedly existed in the past and warrant a brief discussion. '*Marasmus*' is a severe wasting disease resulting from a grossly inadequate diet (Griffith, 1919). The condition is due to a deficiency in both protein and calories causing muscle wasting, anaemia, subcutaneous fat loss and growth retardation. Children with marasmus may survive for up to eight months, and in the past commonly developed bedsores on the occipital bone as a result of their immobility (Holt, 1950). In

the nineteenth century, marasmus was associated with infants living in the unhygienic conditions of cities and infant institutions, but was rarely seen in the countryside or in the upper classes (Griffith, 1919). Today, the condition is more common in bottle-fed babies who come into contact with infected milk and develop diarrhoea. The condition is chronic in areas of low socio-economic status and is associated with iron deficiency anaemia. There is a high mortality rate as children suffer from gastroenteritis and diarrhoea, and are susceptible to infections such as tuberculosis, measles and whooping cough (Griffith, 1919; Sheldon, 1943).

'Kwashiorkor' is a wasting condition resulting from a low protein but a high calorie or starchy diet (protein-energy undernutrition). The condition has a rapid onset brought about by diarrhoea and fever and is usually fatal (Scrimshaw *et al.*, 1959). Children with kwashiorkor are usually small for their age, irritable and susceptible to infection. Kwashiorkor is characterised by chubby cheeks and a swollen abdomen, and Wells (1975) suggested that several representations of this condition are evident in ancient sculptures from Mexico and India. In their milder forms, marasmus and kwashiorkor are characterised by poor physical growth (Martorell, 1980), and in skeletal samples may be suspected in individuals who are small for their age cohort. In addition, Sweeney and colleagues (1971) have shown that enamel hypoplasias on the deciduous incisors are present in 73% of Guatemalan children hospitalised with the conditions.

4.4.2 Rickets (Vitamin D Deficiency)

4.4.2.1 Aetiology and Pathogenesis

The clinical changes associated with rickets were first described by Glisson in 1650 (Griffith, 1919). Rickets affects all the systems of the body and results from a deficiency in cholecalciferol, commonly known as vitamin D_3. Precursors of the vitamin can be found in plants and grains (ergosterol) but 90% of the hormone is produced by the skin's dermal cells from ultraviolet light (Aufderheide and Rodriguez-Martin, 1998). Vitamin D is needed to absorb and mobilise calcium and phosphorus from previously formed bone, and to promote maturation and mineralisation of the organic matrix (Resnick and Niwayama, 1988). Rickets usually occurs during rapid growth periods, and therefore it is common between the ages of 1-2 years and may reoccur around the age of six (Harris, 1933). Paradoxically, if a child is suffering from marasmus and has retarded growth, the signs and symptoms of rickets will not appear until the marasmus has been cured (Griffith, 1919). If the condition is not treated the child becomes susceptible to secondary infections and may die.

Vitamin D is passed to the foetus via the placenta *in utero* and can protect the infant for up to four months after birth (Sheldon, 1943). Hence, premature babies are susceptible to the condition as they have missed out on the last two months of foetal life when the storage of calcium salts occurs (Arneil, 1973). In addition, low birth weight and twinned babies are also susceptible due to their greater need of calcium and phosphorus as the result of 'catch-up' growth after birth. Chick (1976) reported that mothers of rachitic children had lower levels of the vitamin in their breastmilk than those without, and suggested that the body first looked after its own requirements before altering levels in the milk.

Some cases of rickets are the result of defects in the individual's metabolism and cannot be considered the result of cultural or environmental change. Longstanding renal failure, chronic acidosis, hepatic or pancreatic disease can lead to rickets but were probably not compatible with life in the past (Caffey, 1978). Vitamin D resistant rickets (refractory rickets) is a congenital condition requiring massive doses of vitamin D for treatment and can lead to dwarfism in adults (Holt, 1950). Abnormal bowing of the long bones has been reported in poorly reduced midshaft fractures, and bowing due to faulty foetal positioning can persist for up to two years after birth (Borden IV, 1974; Caffey, 1978).

4.4.2.2 Skeletal Changes of Rickets

Figure 4.5 illustrates the distribution of rachitic lesions in the non-adult skeleton. The manifestation of rickets on the skeleton is related to the general nutrition of the child, the age at which the condition develops and their mobility. In the undernourished child, the cortices are thinned and marrow spaces enlarged with general atrophy, and other conditions, such as scurvy, may be present. In a well-nourished child, rickets results in thickened cortices and a reduced medullary cavity (Stuart-Macadam, 1989). These children are described as having 'plump bones' as the long bones, ilia and scapulae thicken under the periosteum, as a result of the deposition of '*several layers of friable, spongy vascular tissue, with large vascular spaces*' (Griffith, 1919: 588).

In rickets the long bones are susceptible to fracture, and there is a general retardation of growth and osteopenia with excessive proliferation of cartilage at the metaphyseal ends of the bone that fails to be calcified. The metaphyseal ends of the long bones become widened and concave as a

result of weight bearing (‘*trumpeting*’) (Figure 4.6), the cortical bone is thinned and the bones develop frayed edges, resembling ‘bristles of a brush’ on radiograph (Caffey, 1978).

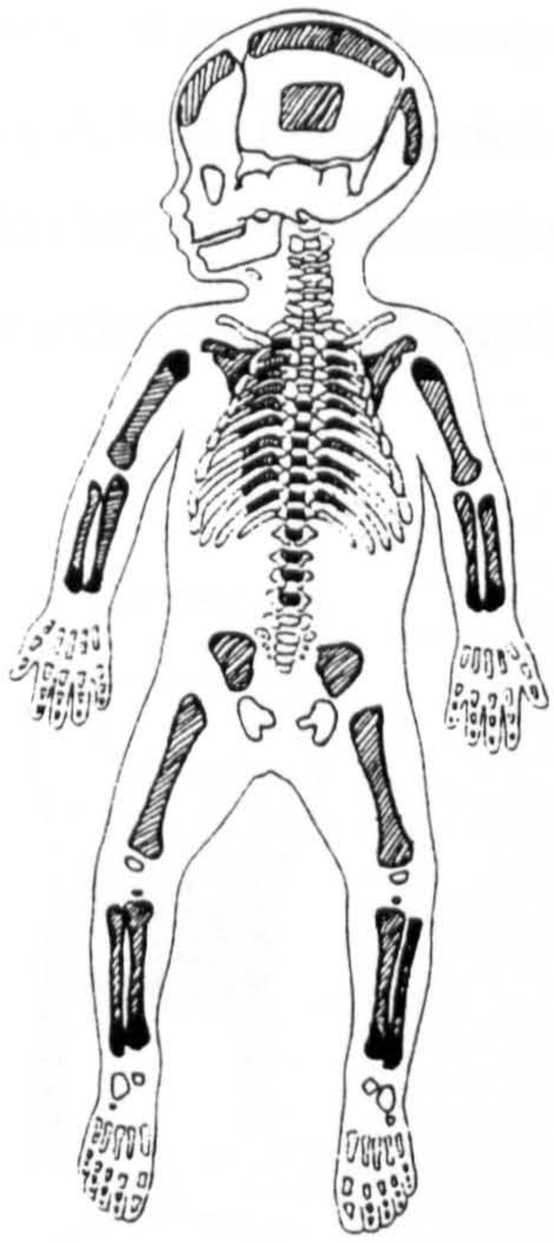
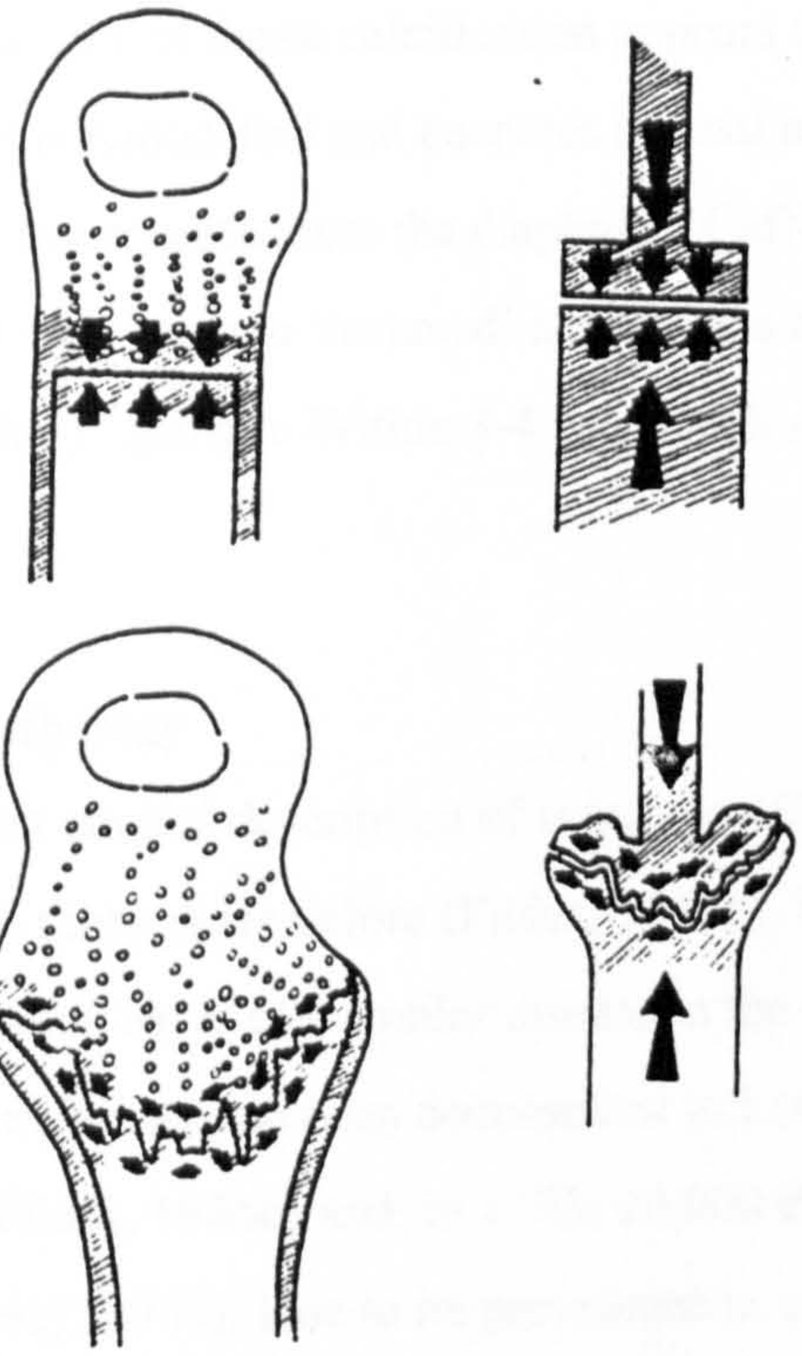


Figure 4.5 Distribution of lesions associated with rickets in the non-adult (black denotes areas most severely affected).

Macroscopically, the characteristic smooth epiphyseal surface of the metaphysis is lost and, on X-ray, the distal end of the bone, made up of uncalcified cartilage, appears to be invisible. Cupping deformities are also present on the costal ends of the ribs and are known as the ‘*rachitic rosary*’ (Resnick and Niwayama, 1988). Other deformities of the rib cage result in a ‘*pigeon chest*’ where the costochondral junctions are weak and inwardly displaced (Caffey, 1978).

The severity of the deformities depend on the rate of growth of the bone at the time the disease commences (Harris, 1931). The wrist (distal ulna and radius) is usually the first area where changes are visible radiographically, but one of the earliest signs of the disease is in the skull. The cranium becomes squared (‘*craniotabes*’) on the side the infant lies, as a result of pressure on soft bone (Mankin, 1974). In addition, immature new bone is deposited on the external table of the

skull, mimicking porotic hyperostosis (see Figure 3.4), and the anterior fontanelle may remain open until the third year. Enamel hypoplasias are common and dental development is delayed (Sheldon, 1943) which is a matter of concern when trying to estimate the age of these individuals in a skeletal sample. For example, Holt (1909) carried out a survey on dental eruption in 150 rachitic children from a London hospital, and found that in 20 cases the dentition did not erupt until the twelfth month, and in eight cases dental eruption was delayed for up to fifteen months.



In the normal epiphyseal plate, growth in length is produced by epiphyseal proliferation on the calcified cartilage and a rigid ossified metaphysis (above). In the rachitic plate, the softened cartilage, metaphysis and calcified zone collapse and spread as a result of the distorted growth pressure and external forces (below).

Figure 4.6 Representation of the metaphyseal ends of long bones as a result of rickets (after Mankin, 1974: 111*).*

It may be possible to suggest an age of onset for the condition; bowing of the arms where the humeral head is bent medially and inferiorly suggests the child may have been crawling at the time of onset. An abnormal antero-lateral concavity of the femoral shaft and a bowed and antero-posteriorly flattened fibula, indicate that the child had begun to walk (Stuart-Macadam, 1988). However, children with rickets have a habit of sitting cross-legged and this posture may contribute

to the deformities (Holt, 1909). Anterior curvature of the tibia is considered to be a late sign of the disease and, in all the affected bones, bowing is a result of weight bearing and tendinous strain on a weakened growth plate (Caffey, 1978). Children who continue to maintain good muscle tone and remain mobile during the course of the disease have been shown to develop more severe bowing, cupping and flaring deformities than those with poor muscle tone who remain stationary (Stuart-Macadam, 1988). If the child is not treated and survives over the age of four years, stunting and deformities can persist (Sheldon, 1943).

Within 2-3 days of treatment, a zone of dense calcification appears at the end of the bone. The radiolucent rachitic metaphysis is remodelled and becomes normal in appearance and the calcification band is gradually incorporated into the diaphysis (Caffey, 1978; Harris, 1931). The gross enlargements at the joint ends become 'trimmed' and there is a period of intense remodelling above normal, similar to 'catch-up' growth. Within 3-4 months all signs of the disease may have disappeared (Harris, 1931).

4.4.2.3 History and Palaeopathology

When Glisson provided the first clinical description of rickets in 1650 it was considered a new disease that had only appeared thirty years before (Fildes, 1986b). However, as early as the first century AD, Soranus had reported cases of a similar disease in the urban centre of Rome (Foote, 1927). In 1634, 14 deaths from rickets had been documented in London but, by 1659, as many as 1,598 deaths were recorded (Fildes, 1986b) and, in 1773, 20,000 children were reported to have been affected in the city (Harvey, 1968). Due to its prevalence in urban areas, rickets became known as a disease of air pollution and was so rampant in Britain that Whistler referred to it as the 'English Disease' (Mankin, 1974). In 1889, the British Medical Association carried out a survey on the distribution of rickets in Britain and found the disease to be prominent in the industrial and mining areas. As their survey moved further away from the urban centres, the frequency of the disease declined (Hardy, 1992). However, Britain was not the only place where the condition became endemic. During the 1900's Morse diagnosed rickets in 79.5% of 400 Boston infants and, in Germany, 89% of Dresden infants aged between 2-4 years had the disease (Griffith, 1919). In 1890, Palm commented that the greater prevalence of rickets in Northern Europe was the result of poor levels of sunlight, exacerbated by the smoky atmosphere of the Industrialised cities (cited in Chick, 1976).

In 1919 Mellanby, a British nutritionist, noted the effective use of cod liver oil in treating the signs and symptoms of rickets, and hence proved it to be a deficiency disease (cited in Resnick and Niwayama, 1988). This finding fitted with previous observations in urban areas, where Jewish children appeared to be unaffected by the disease, and their diet, made up predominantly of fried fish, may have provided them with the protection they needed (Hardy, 1992). The quality of breastmilk was another factor thought to contribute to the incidence of the disease. Parry (1872) claimed that the idle lifestyle of the rich, and the poor living conditions and excessive exercise in the poor, resulted in low-quality breastmilk. The lower prevalence of the disease in middle-class children was, Parry argued, due to good quality breastmilk produced by a balanced diet and appropriate level of exercise. In addition, these poorer children could play outside where they were exposed to ultraviolet light, and they drank cow's milk, which was considered a food for invalids and infants by the rich (Fildes, 1986b). Rickets was closely associated with whooping cough and measles in the nineteenth century and this contributed to the high mortality rates from the disease in the lower classes where it was difficult to protect the children from frequent epidemics (Hardy, 1992).

Despite the endemic nature of rickets in the Industrial period, the diagnosis of this condition has, until recently, been neglected, with older studies recording the prevalence of enamel hypoplasias as evidence for the disease (Wells, 1975). Nevertheless, diagnostic techniques are becoming more sophisticated and Ortner and Mays (1998) have recently described eight cases of rickets in rural Wharram Percy; one case was also discovered at Jewbury in York and at St. Helen-on-the-Walls (Dawes and Magilton, 1980; Lilley *et al.*, 1994), and around 20 cases were reported at Christ Church Spitalfields in London (Molleson and Cox, 1993). The early changes of rickets are subtle and usually begin with an expansion and fraying of the rib ends and distal radius. The extreme bowing deformities are later manifestations, and occur when the child applies weight to the affected limbs. However, the disease can render children immobile and, as they are susceptible to respiratory and gastrointestinal infections as a result of the disease, they may not live long enough to develop the most obvious signs. These factors may explain why so few cases of the condition have been reported in the archaeological record.

4.4.3 Infantile Scurvy (Vitamin C Deficiency)

4.4.3.1 Aetiology and Pathogenesis

Scurvy results from a dietary deficiency of ascorbic acid or vitamin C, present in wide variety of fruits and green vegetables. Today, infantile scurvy is present in children fed formulas containing pasteurised or boiled milk, a process that destroys the small amounts of vitamin C contained within it. The disease is clinically recognisable when the vitamin has been deficient for 4-10 months, or once birth stores have been depleted (Ortner and Putschar, 1985). Hence, scurvy is most common between six months and two years of age and, as with rickets, premature, low birth weight babies and twins are susceptible (Griffith, 1919). Vitamin C is necessary for the formation of collagen, the major structural protein of the body, which forms the basis of connective tissues for the skin, cartilage and bone, and it also protects and regulates the biological processes of other enzymes (Stuart-Macadam, 1989). Haemorrhage of the mucous lining of the gums produces bleeding around already erupted teeth, and bleeding due to capillary haemorrhage in the most rapidly growing areas of the skeleton occurs during the first two years of life and affects the cranial vault, orbits and ribs. Anaemia results from abnormal erythroblastic areas, where grey marrow replaces red bone marrow at the end of the long bones, leading to arrested growth.

4.4.3.2 Skeletal Changes of Scurvy

In 1883, Barlow discussed rickets and scurvy both as individual diseases and as conditions that frequently occurred together. For this reason scurvy was not recognised as a childhood condition for many years, as the changes associated with rickets could be identified early in the disease, whereas those resulting from scurvy only appeared in advanced cases and were masked by rickets (Barlow, 1935; Follis and Park, 1952). The skeletal manifestation of scurvy results from the primary effect of vitamin C deficiency and the secondary effects of trauma to weakened bone.

Figure 4.7 illustrates the distribution of lesions in the non-adult skeleton. At the metaphyseal end of the bone, cartilage cells become reduced in size and number and are thickened by continuous layers of osteoid. This heavily calcified band is weak and made up of irregular trabeculae which are susceptible to fracture. Spurs result from the extension of this heavily calcified layer and periosteal deposition. Directly preceding this band is an area of thinned trabeculae, appearing as a radiolucent band or '*scurvy line*' on radiographs (Caffey, 1978). Harris (1933) believed that the width of the zone corresponded to the length of time the child had had the condition. Once healing begins, the scurvy line disappears and the band of dense calcification is only recognised as a

Harris line. In the epiphyses, a shell of heavily calcified bone surrounds a rarefied ossification centre. Radiographs of the epiphyses can aid in the diagnosis of scurvy particularly when the condition has healed, as the changes in the epiphyses are often still present some years after the other signs of the condition have been remodelled (Caffey, 1978). In some cases, the distal femur may become cupped, encompassing the epiphysis which fuses to the end of the bone preventing any further longitudinal growth (*ibid.*, 1978).

Sub-periosteal haemorrhage is most frequently seen at the ends of the femur, tibia and humerus, but may be present on the entire length of the diaphysis (Caffey, 1978). Harris (1933) carried out a survey of lesions in scorbutic children under three years of age and found that evidence of haemorrhage on the skeletal system occurred in 93% of the cases and, in the orbits, 37% of cases showed either bilateral or unilateral lesions. Haemorrhage of the orbits is recognised in the palaeopathological record as deposits of new bone on the superior orbital margin. It has been suggested that endocranial lesions may be the result of slow haemorrhage of the dura as a result of scurvy (Ortner, *pers. comm.*) and histological analysis has been employed to try and distinguish this condition from anaemic bone reaction on the skull (Carli-Thiele, 1995). With healing, the new bone formed as a result of haemorrhage remodels resulting in a slight thickening of the affected bones.

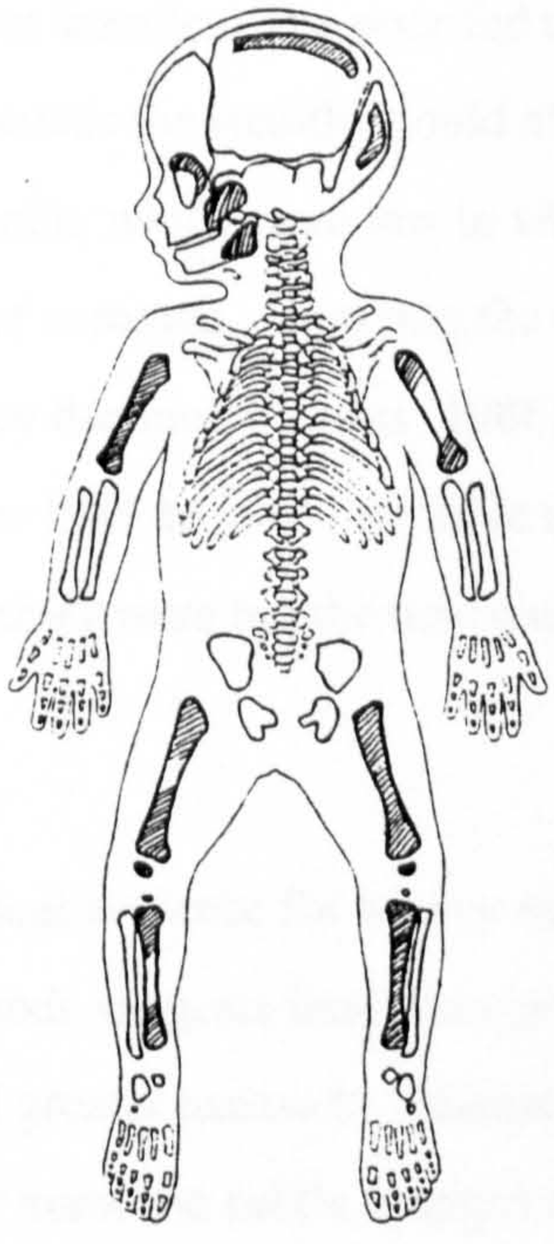


Figure 4.7. Distribution of lesions indicative of scurvy on the non-adult skeleton

The main diagnostic feature of scurvy on the skeleton is the bilateral porosity evident on the bones of the skull. Ortner and Ericksen (1997) have identified lesions at the insertions of the temporalis muscles, the posterior maxilla and the greater wings of the sphenoid; the latter lesion is now considered to be pathogonomic of the condition (Ortner *et al.*, 1999). The porosity is thought to occur as extravasated blood, resulting from venous rupture, causes inflammation and new blood vessels to penetrate the cortex. Although anaemia may result from this condition, porosity of the orbital roof (cribra orbitalia) may, in some cases, represent haemorrhage rather than marrow hyperplasia (*ibid.*, 1999).

4.4.3.3 History and Palaeopathology

Despite Glisson's mention of the disease in 1950 in relation to rickets in children, infantile scurvy was not described as a clinical entity until the nineteenth century when, in 1883, Barlow set out the clinical and pathological signs of the disease (Barlow, 1935). However, the cause of the condition was not discovered until 1919 (Wilson, 1975). Before the sixteenth century, scurvy was common in soldiers and sailors on long sea voyages, but was not recognised as a childhood disease until the rise of urbanisation. Wilson (1975) suggests that, although present, infantile scurvy may have gone unnoticed because swollen, bleeding gums, readily identified as scurvy in adults, did not occur in children before their teeth had erupted. In the eighteenth century, scurvy was common in the wealthiest families. The poor fed their children on a diet of potatoes and gravy, rich in vitamin C, whereas the wealthy could afford the fashionable formulas including pasteurised and condensed milk, which were low in vitamin C (Harris, 1933; Lomax, 1986). As market gardens became more common, supplying the cities with fresh produce, and the potato became more popular, scurvy declined (Lomax, 1986). However, artificial infant feeding practices still had an impact and, from 1945 to 1965, infantile scurvy was common in the lower income families in Canada where babies were fed the now cheaper condensed milk (Stuart-Macadam, 1989).

The lack of palaeopathological evidence for scurvy may be due to its rarity in the past, and in the later and post-medieval periods suggests that many people were too poor to buy fashionable formulas and therefore, had greater access to Vitamin C in their diet. However, it is also likely that co-existing rickets may mask the subtle changes of scurvy in the skeleton. In 1997, Ortner and Ericksen reported six cases of scurvy from Peru, two cases from Beirut and one from Norway. In a following study of 363 skulls from different areas of Peru they found the disease to

be more common in 7-12 year olds. This unusually late prevalence of the disease, they suggest, results from a bias in the sample, which has very few younger children (Ortner *et al.*, 1999). Schultz (1989) diagnosed scurvy in 13.8% of infants from Bronze Age Europe and Carli-Thiele (1995) claimed that scurvy was evident in 40% of a Neolithic population, after histiological examination. Roberts (1987) described a case in an Iron Age child from Worcestershire displaying new bone formation around a deciduous maxillary molar, the tibia and in the orbits and Wells (1975) found evidence in 2% (*n*=7) of Anglo-Saxon skeletons from East Anglia.

4.5 Summary

A review of the childhood diseases that can be identified on non-adult skeletons suggests that as societies became more urbanised, respiratory infections such as sinusitis and tuberculosis increased. Changing attitudes towards infant feeding with the rise of female labour, and air pollution in the industrialised cities of the eighteenth and nineteenth centuries, also contributed to a high prevalence of metabolic disorders such as rickets and scurvy, which had hitherto gone unrecognised as childhood disorders. Non-specific infections such as periostitis and endocranial lesions are also useful measures of child health in the past and will become increasingly important as research into their causative factors progresses.

CHAPTER FIVE

MATERIALS: HISTORICAL AND ARCHAEOLOGICAL BACKGROUND TO THE SITES

5.1 INTRODUCTION

The sites used in this study were chosen for their geographical location (Figure 5.1), large non-adult sample size and contrasting settlement character. Raunds Furnells in Northamptonshire is the earliest of the settlements, with a cemetery dating between the tenth to twelfth centuries. The inhabitants of this Anglo-Saxon site would have practised subsistence agriculture. The parish of St. Helen-on-the-Walls, based in pre-industrial York, dates from the eleventh to sixteenth centuries and was contemporaneous with Wharram Percy, a deserted medieval village in the Yorkshire Wolds (c. AD 900-1500). Although the inhabitants at Wharram Percy would have been living in a rural environment, they would also have had contact with the urban centre of York. Finally, non-adults excavated from the crypt of Christ Church Spitalfields, in London (AD 1729-1859) were chosen to represent the post-medieval sample. This chapter will outline the archaeological and historical contexts of the sites and test for any under-representation of the non-adult material.

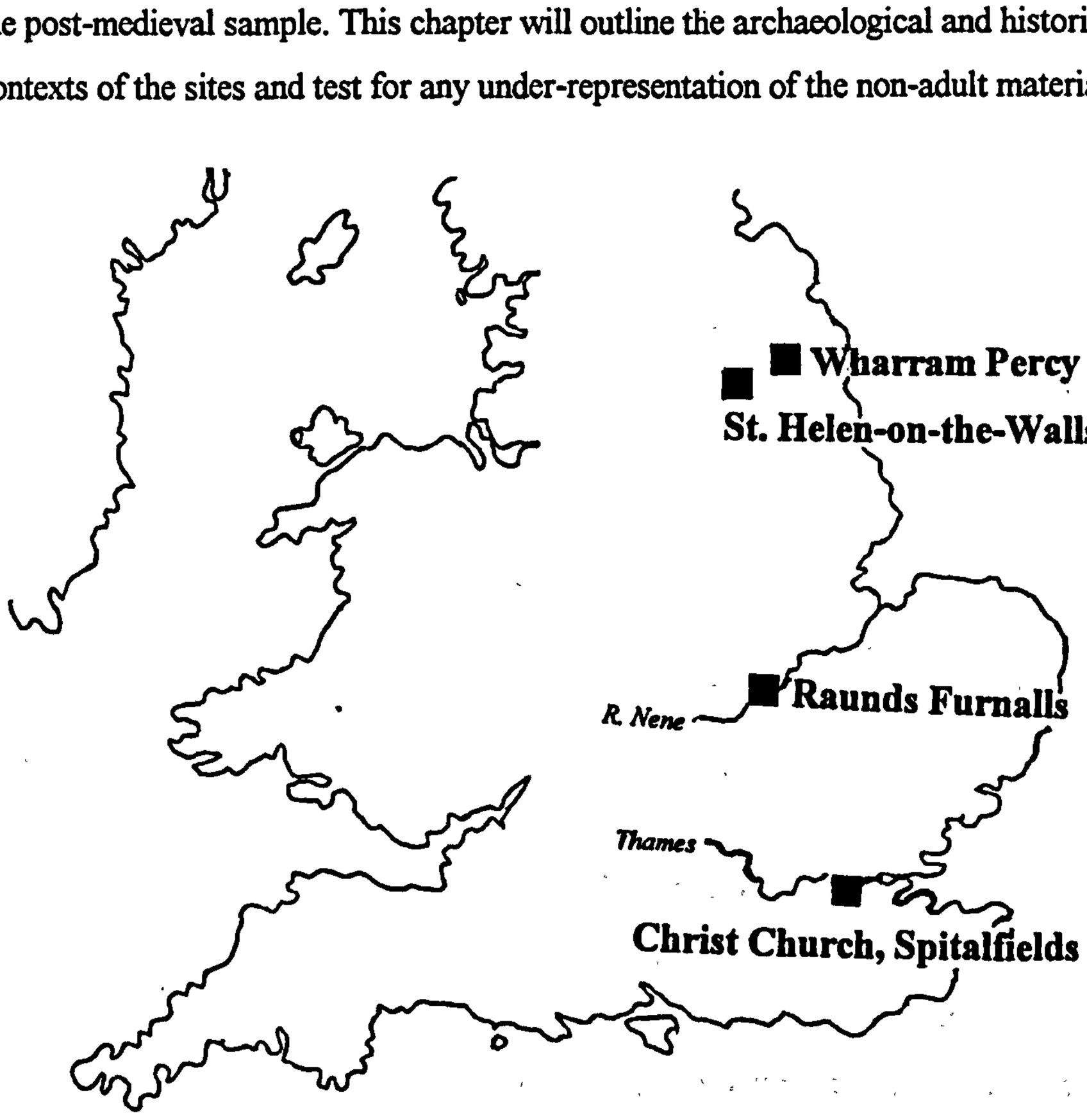


Figure 5.1. Geographical location of the sites

5.2. RAUNDS FURNELLS, NORTHAMPTONSHIRE

5.2.1. HISTORICAL BACKGROUND

Raunds is situated in a valley on the eastern side of the River Nene, in the county of Northamptonshire. The village was first mentioned in AD 980 in the Anglo-Saxon Chronicle as '*Randa*', but archaeological evidence suggests that the community had existed since at least AD 850 (Boddington, 1996). In the Late Saxon period (AD 850-1100), Raunds was the most dominant and largest settlement of four hamlets including Stanwick, Hargrave and Ringstead (Figure 5.2), and was continually occupied from the sixth to late fifteenth centuries. In AD 1066, Raunds was held by two manors, that of Burgred and Gytha, and had six dependent villages (Cadman and Foard, 1984). It is thought that the manor Burgred was originally at the site of Furnells, to the north of the present village, and that this was the primary manor of the area until the medieval period, when Burystead, held by Gytha, became prominent.

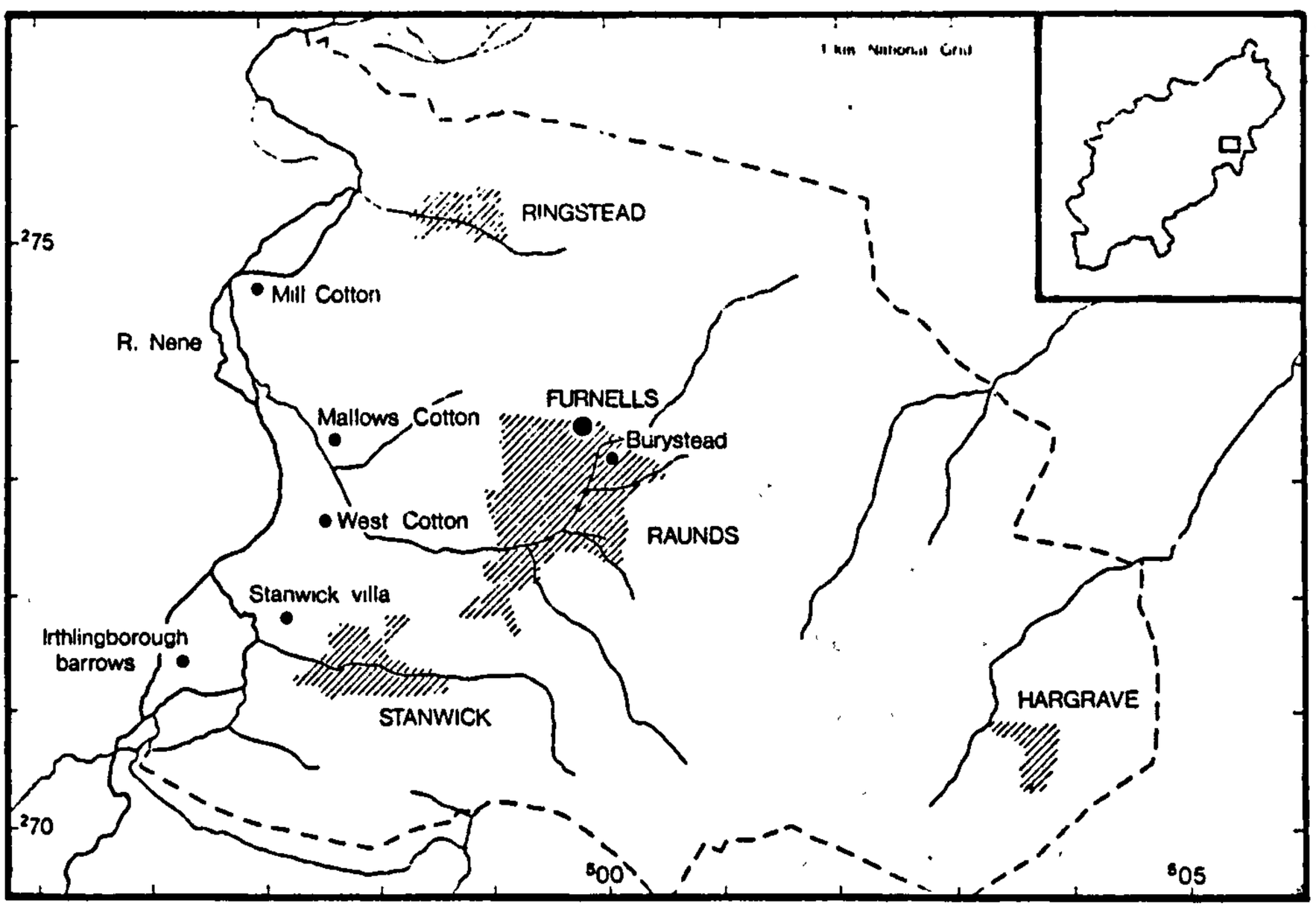


Figure 5.2. Location of Raunds Furnells (Boddington, 1996: 1)

The discovery of elaborately carved stone coffin lids in some graves at Raunds Furnells has led to suggestions that the manor was occupied by a wealthy bailiff and his family (Boddington and Cadman, 1981). In addition, the size of the church indicates that 30-40 people inhabited Furnells at any one time. The type of burials in the churchyard suggest a

range of social divisions consistent with those identified at the manor of Burgred in 1066, which included four slaves, four villagers, six small holders and 16 dependants.

Owing to the perishable nature of Anglo-Saxon buildings and artefacts, little is known about the Late Saxon settlement and their crafts and industries. However, individuals at Raunds Furnells were likely to have been engaged in subsistence agriculture, and have had use of the meadows and woodland within the surrounding area. Most of the available woodland had been turned into arable land by 1086 (Cadman and Foard, 1984). Environmental evidence from neighbouring villages showed that wheat, barley, rye and flax were being grown (Campbell, 1994), and it is suggested that similar crops were being cultivated at Raunds Furnells. These crops would have been used to produce bread, porridge, beer, clothing and fodder for animals.

The houses were probably consistent with the period: timber-framed rectangular buildings with stone or clay floors. There would have been a living quarter at one end and perhaps a byre at the other. Close contact with cattle would have left the occupants susceptible to infections (zoonoses), allergies and parasitic infestation, but this Late Anglo-Saxon settlement pre-dates any of the major industrial activity that developed in England during the thirteenth century.

5.2.2. Archaeological Evidence

Furnells is situated to the north of the village of Raunds (Figure 5.2). The site was excavated by the Northamptonshire County Council Archaeological Unit between 1977 and 1979 when a manor, church and churchyard were uncovered (Boddington and Cadman, 1981). Excavations revealed that the site initially contained timber buildings organised in a rectangular enclosure. By the eleventh century, they had merged to form a single manor house. A church was evident at the site from the early tenth century and, during the next 200 years, was enlarged and a graveyard added. In the twelfth century, the church and churchyard at Raunds Furnells fell into secular use, the churchyard was abandoned and the church building incorporated with the manor house (Cadman and Foard, 1984).

The individuals from the churchyard were probably interred during the tenth to twelfth centuries and were dependants of the manor. Most of the burials were in the south and east of the cemetery and there was a concentration of infants on the south side of the church wall, suggesting they were placed under the '*eaves-drip*' where, according to canon law, children

could be indirectly baptised (Boddington, 1996). The low incidence of inter-cutting graves suggests that permanent markers were being used (Figure 5.3). However, during the enlargement of the church, grave mounds, markers and crosses were destroyed and may have resulted in the loss of some of the burials (*ibid.*, 1996).

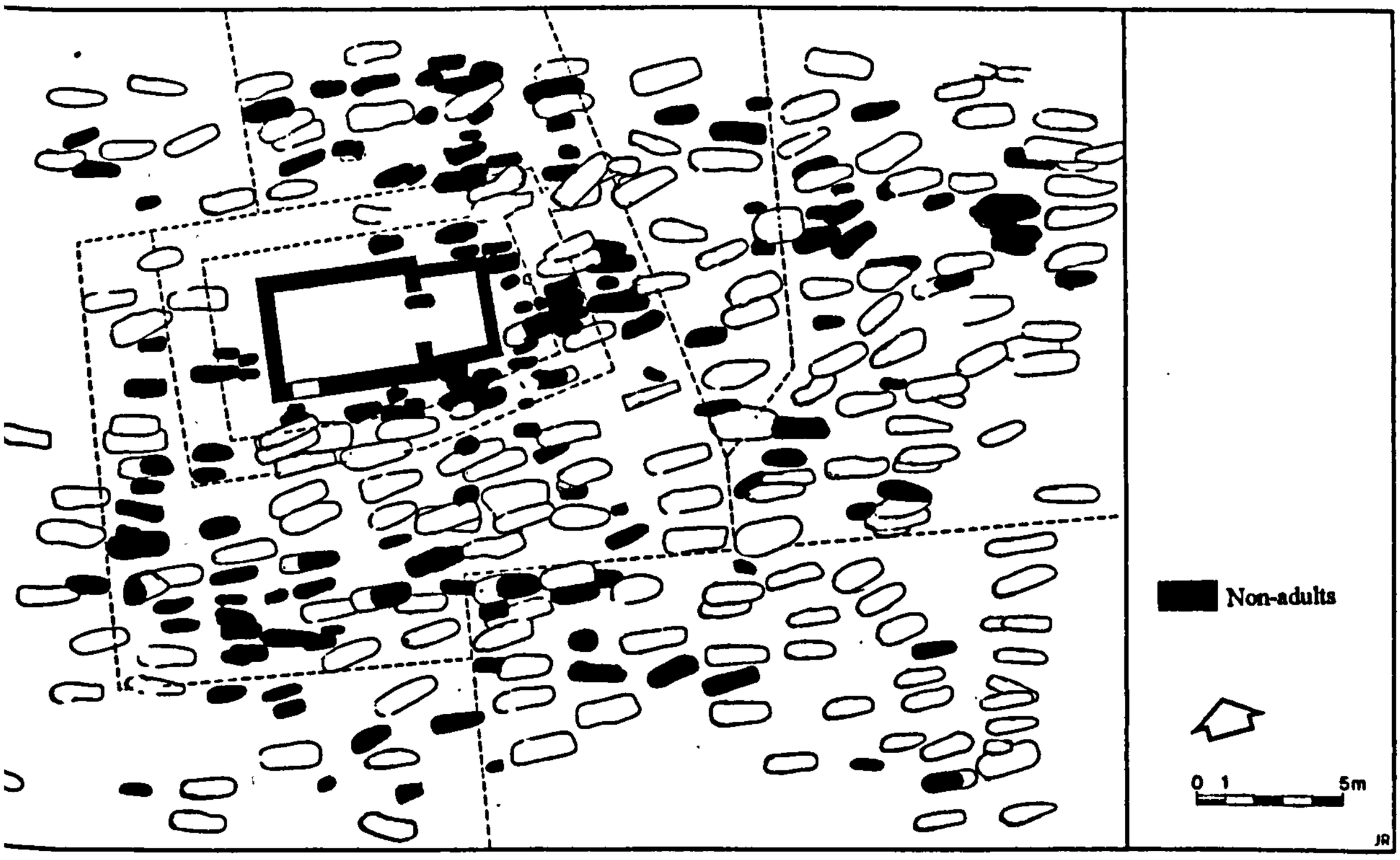


Figure 5.3. Plan of Raunds Furnells cemetery, showing burials of the non-adults and the location of the earliest church (Boddington, 1996).

Three hundred and sixty-three skeletons were recovered in 1979 but only 346 were available for this study. In the initial report, Powell (1996) classified non-adults as those aged between 0-17 years and her sample consisted of 193 (53%) adults, 149 (41%) non-adults and 21 (6%) preterm individuals. The present study included 126 non-adult and 23 preterm skeletons. Twenty-one non-adults were not located and may have been among the skeletons destroyed during an explosion next to the storage area in the early 1980 s. The preservation of the surviving skeletons was generally poor and many of the non-adult remains were fragmentary as a result of long-term storage and usage, and the earlier explosion. However, it was clear that excavation of the non-adult remains had been carefully carried out, and the preservation of the foetal remains in particular was excellent.

(See Appendix XI, Table A for number of non-adult individuals reported previously and the number in the present study. Table B summarises the number of individuals aged by each techniques for each site.)

5.3 St. Helen-on-the-Walls, York

5.3.1. Historical Background

'The city of York is the capital of all the Northumbrian people. ...It boasts...a huge population, of not less than thirty thousand of both sexes, excluding children. It is crammed with the merchandise-too rich to describe here-of traders who come from all parts...'

Description of York in c.1000, by the monk of Ramsey (cited in Ottaway, 1992: *146*).

Medieval York was at its height in wealth and influence between the eleventh and fifteenth centuries. It was the focus of social, political and religious activities for the North and ranked second in the kingdom only to London (Andrews, 1984). The parish church of St. Helen in Aldwark was first mentioned in the twelfth century as one of three churches in York dedicated to Constantine's mother. The names used to distinguish the church from the others (i.e. St. Helen in Aldward, St. Elene in Werkdyke and St. Ellen *ad muros*) suggests that the church had earlier origins, perhaps in the tenth century (Palliser, 1980). Major redevelopment of the church in the 1420 s is thought to have led to its rededication by the Bishop of Dromore in 1424 but, despite this apparent display of wealth, St. Helen-on-the-Walls was regarded as a poor parish.

Between 1420 and 1434, St. Helen was one of the parishes placed in the lowest tax bracket, paying £3 a year as opposed to the £14 paid by the richer parishes (Palliser, 1980). In 1548, the church was coupled with neighbouring St. Cuthbert as the result of an Act of Parliament, which stated that poor parishes should be merged. In 1550, the land was boughtt in a private sale and 25 years later the building lay in ruins. The site was forgotten by 1610 and was omitted from a plan of York (*ibid.*, 1980).

Aldwark was a street of mixed wealth, with St. Leonard's hospital and the College of Vicar's Choral of the Minster at one end, and "*dilapidated timber-framed cottages crowding along the street frontage*" at the other (*ibid.*, 1980: 58). In the thirteenth century, property owners ranged from craftsmen of the fine leather industry and the Yorkshire Knightly class, to dyers, chandlers, brewers, tanners, masons, carpenters and fullers (Grauer, 1989). These individuals probably found occupation within the foundry, apothecary, tanning industry and brewery, situated in the neighbouring parish of Bedern. As the result of the Black Death, by the fourteenth century property prices had fallen to some of the lowest in the city. There was a shift in the residence of this area, from mostly male

craftsmen to females, some suspected to have been working as part-time prostitutes (Hall *et al.*, 1988).

Excavations at Aldwark revealed that fences and walls built on baulks separated the houses. They were generally timber-framed with tile roofs and may have been divided into separate rooms with clay lined floors. There was also evidence for cobbled courtyards (Hall *et al.*, 1988). Although there is little direct evidence for living conditions in Aldwark, it is known the buildings had been left to fall into disrepair.

The size of the population, extensive settlement and specialised industries would have led to overcrowding, environmental pollution and a build-up of domestic and industrial refuse in York. Effective methods for the disposal of the refuse, and a regular supply of food and clean water would have been needed to ensure the health of the population (Addyman, 1989). However, evidence from the medieval city of Winchester suggests that these factors posed a serious health risk in medieval towns. Sewage disposal was a problem, cess-pits seeped and undermined neighbouring houses, and numerous edicts indicate that cattle, pigs and poultry roamed the streets, raiding vegetable markets and occasionally attacking small children (Keene, 1982). Butchers dumped offal in the streets and, due to complaints, were regularly ordered to transport the waste to the river Ouse, the main water supply for the city. In 1332, Edward III described York as the '*filthiest, most foul smelling city in the kingdom*' (Keene, 1982: 28).

5.3.2. Archaeological Evidence

The small parish church and associated cemetery of St. Helen-on-the-Walls (hereafter St. Helen) was first excavated in 1972 by the York Archaeological Trust, under the site of the Ebor Brewery. Initially, the church was a small rectangular structure, but during its lifetime four phases of improvements were made, resulting in the addition of a chancel and a bellcote (Magilton, 1980). As is common with urban excavations, the extent of the cemetery was not fully determined due to existing structures, and many of the burials in the south were destroyed by the Victorian brewery well. The Merchant Taylor's Property wall is thought to represent the northern limit of the cemetery, with the west being marked by a medieval ditch, or '*quensdyke*' (Figure 5.4). The north-west corner of the site remains unexcavated and in all, only two thirds of the original cemetery area is believed to have been uncovered (Dawes and Magilton, 1980). The cemetery was densely packed, utilising the north side of the church, usually reserved for suicides and unbaptised infants. In addition, the density of

the graves and a lack of permanent grave markers resulted in extensive intercutting, and subsequent damage to the preceding burials (Magilton, 1980).

The human remains probably represent parishioners interred in the cemetery between the years AD 950-1550. There were three charnel pits and 1186 separate deposits, 65% (*n*=1041) of which were identified as individual burials. There was a striking under-enumeration of non-adults and foetuses in this sample and, although there was some evidence of a clustering of infants and young adults in the southern corner of the cemetery, no specific area seems to have been set aside for them. Magilton (1980) suggested that some non-adults may lie in the unexcavated north east side of the graveyard.

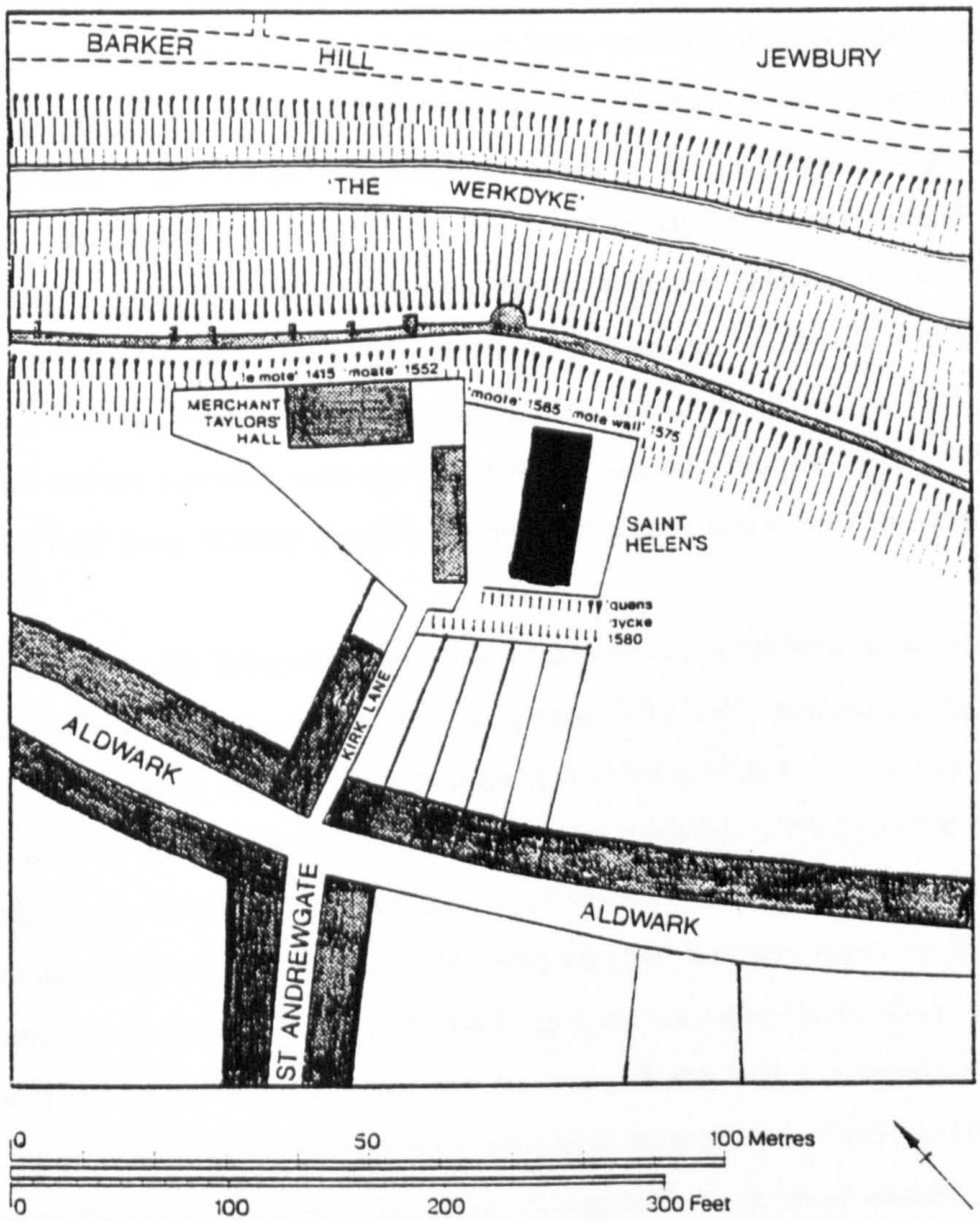


Figure 5.4. Location of the church and churchyard of St. Helen-on-the-Walls (Magilton, 1980:4).

In the initial report, skeletons were categorised as non-adults if they were below the age of 17 years (0-16 years). Here, 724 (69.5%) were recorded as adults, 284 (27.3%) as non-

adults and 33 as (3.2%) preterm individuals (Dawes and Magilton, 1980). In the present study, there were only 231 non-adults including seven preterm individuals. The large number of unlocated skeletons cannot merely be due to different non-adult age categories and may be, in part, due to the problems of 25 years of storage and curation.

5.4 Wharram Percy, North Yorkshire

5.4.1 Historical Background

Wharram Percy is a deserted medieval settlement situated on a terrace on the west side of the Yorkshire Wolds, 18 miles from the city of York (Bell and Beresford, 1987). Wharram Percy was first mentioned in the Doomsday Book of 1086 and, like Raunds Furnells, consisted of two manors. The Percy family united the manors in 1254. St. Martin's church and it's cemetery, located to the north of Wharram Percy served four other villages from the area; Burdale, Raisthorp, Towthorpe and Thixendale. St. Martin's became a parish church in the twelfth century and subsequent expansions indicated that, at its height, the parish catered for around 30 families (Beresford and Hurst, 1990). By 1323, the population of Wharram Percy had decreased as a result of the Black Death and famines, and in 1334 was ranked one of the lowest in taxable wealth for the area. However, by 1368 the village experienced a revival, possible due the acquisition of the manor by the Percies, and some of the tenements and the watermill were put back into use. Depopulation of the village to make way for open field sheep farming began in the fifteenth century and was complete in 1517.

Little is known about the living conditions at Wharram Percy, as archaeological evidence for the buildings is limited due to complex site stratigraphy. However, Andrew and Milne (1979) suggest that before the fourteenth century villagers lived in one storey, timber-framed long-houses, ranging from 40-80 feet in length, with central doorways and floors of compacted chalk and earth, similar to the ones at Raunds Furnells. The roofs were thatched with tiles lining a smoke vent and the floors were concave, indicating that they had been regularly swept (Andrew and Milne, 1979). Therefore, the build up of debris within the building itself, which would harbour parasites and their vectors, was probably kept to a minimum. In addition, the discovery of only two middens in the area suggests that refuse was collected and spread on the fields as manure. This theory is supported by the large amount of pottery sherds discovered on the fields in the surrounding area (Beresford and Hurst, 1990).

By the fifteenth century, stone buildings had been erected and there was evidence of central hearths. Charcoal, and later coal, were burned to heat the houses, and animals would

probably have remained outside, except in bad weather (Andrew and Milne, 1979). Some of the buildings had an industrial purpose, including lime burning, brewing and smithying, but these practices only operated on a small scale. The lack of a well in the village meant that the villagers would have collected their water from the local stream (Mays, *pers. comm.*). There was environmental evidence for the production of wheat, barley and oats and the bones of sheep, cattle, pig, deer and horses were present at the site, but there was little evidence that the inhabitants hunted in the local area (Milne and Richards, 1992). That they ate fish was supported by carbon stable isotope ratios in their bones (Mays, 1997), and the discovery of marine fish, coal and pottery, indicates that the villagers traded with neighbouring urban areas, such as York (Hall, 1988; Milne and Richards, 1992). Thus, for short periods at least, individuals from Wharram Percy would have been exposed to similar environmental pathogens as their urban counterparts.

5.4.2. ARCHAEOLOGICAL EVIDENCE

The excavation of St. Martin's church and it's churchyard at Wharram Percy was carried out between 1962 and 1978, when 681 articulated skeletons were removed from within the church (n=38) and the surrounding area (Harding, 1996).

Owing to the complex stratigraphy of the site, the period of cemetery use was established using radiocarbon dating (Mays, *pers comm.*). The earliest stone church dated from AD 950 but there was evidence of occupation at Wharram Percy since the Roman period. Burials continued at the site until the early twentieth century (1906) and, hence, the south side of the cemetery was left untouched to avoid disturbing these later interments. The individuals used in this study came from sample areas to the north and west of the church, where burials ceased after desertion of the village in the sixteenth century (Figure 5.5). The exact date at which the burials were placed in the graveyard is not known but it is thought to have been between AD 950-1500 (Beresford and Hurst, 1990).

By 1554, Wharram Percy had been abandoned and the cemetery was used by the neighbouring village of Thixendale. Between the seventeenth and nineteenth centuries, individuals were being placed inside the church nave (CN) (Beresford, 1987) and it is proposed that these were the wealthier members of the community at that time (Mays, 1997). Excavations of the churchyard uncovered the graves of Anglo-Saxon noblemen (including one infant) dating from the eleventh century, medieval priests, as well as other

inhabitants of the village. The sample consisted of 681 skeletons; 303 (44.5%) were non-adults of which 32 (10%) were preterm individuals. The level of preservation was excellent.

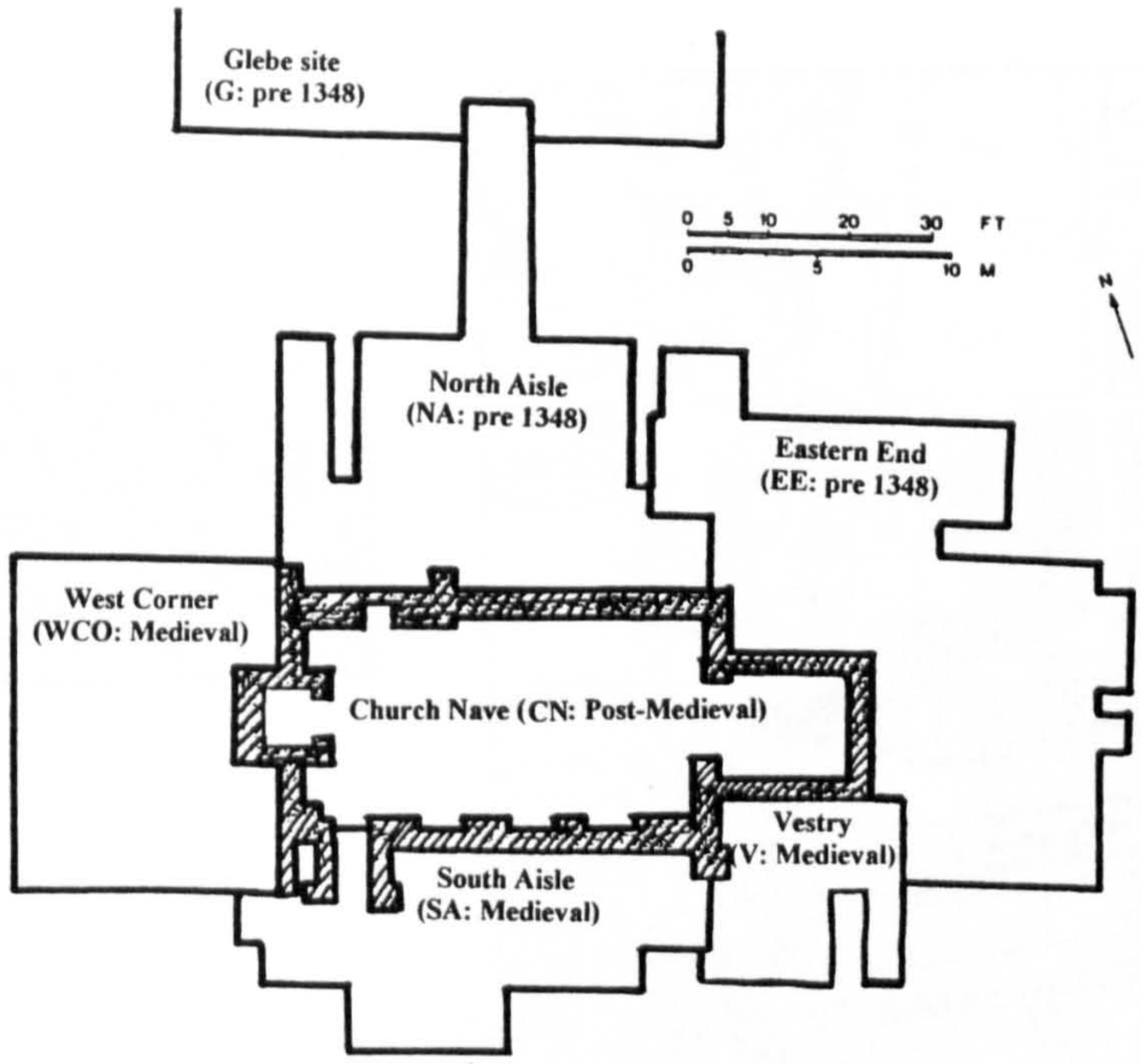


Figure 5.5. The location of the burials at Wharram Percy in relation to the church building. Most of the burials occurred between the tenth-nineteenth centuries (Mays pers. comm.*).*

5.5 Christ Church Spitalfields, London.

5.5.1 Historical Background

Christ Church Spitalfields is located in east London, in the south east corner of Spitalfields fruit and vegetable market (Figure 5.6). The earliest use of this area was as a Roman burial ground, and during the 1530 s it became the favoured area for immigrants and non-conformists to settle (Cox, 1996). In 1572, the Huguenots arrived; they were Protestants escaping religious persecution in France, and they played a major role in the silk industry that became prominent at Spitalfields.

Parish records reveal that the individuals interred in the crypt were the middle to upper class members of the congregation. Their occupations were listed as master craftsmen, gentlemen, surgeons and merchants. Georgian society did not have the sophisticated sewage and water systems of the Victorian age but the people of Spitalfields did benefit from the Lighting and Cleansing Act of 1759 and the Paving Act of 1778 (Cox, 1996). Water was usually supplied through wood and later lead pipes, but was sporadic, and people used storage tanks

to collected rainwater and supplement their supply. In the 1700 s, the population of London was estimated to be 20,000 and, whatever their status, the residents of Spitalfields would have suffered from the effects of overcrowding and inadequate sewage disposal.

Figure 5.7 Modern location of Christ Church, Spitalfields (Reeve and Adams, 1993:4).

5.5.2. ARCHAEOLOGICAL EVIDENCE

Excavations at Christ Church Spitalfields (hereafter Spitalfields) were carried out between 1984-1989, to enable the crypt to be used as an alcoholic rehabilitation centre (Reeve and Adams, 1993). This Anglican church was one of three built in the area during 1715 and, when it was completed in 1730, it was lawful for burials to be placed inside the church. The vaults extended below the entire length of the building and were divided into smaller cells representing private vaults. In 1813, new vaults were added to cope with the rising number of burials until 1859, when the church and churchyard were closed. The ventilation shafts leading from the crypt were bricked up and soil and charcoal deposited on the graves. During the First World War, some of the coffins were removed from their original position in order to use the vault as an air-raid shelter.

From records of the named sample, Cox (1996) estimated that of those interred in the crypt, 38% were from Spitalfields, 39% from neighbouring parishes and 22% from other areas of London; only 1% were estimated to have come from outside the city. The children in the crypt were likely to have been born in Spitalfields but their under-enumeration may be

explained by the practice of sending children to wet-nurses in the countryside. The use of wet-nurses was at its height during the seventeenth and eighteenth centuries and, if the child died whilst being breastfed, they would have been buried in the parish of their wet-nurse rather than being returned home (Fildes, 1988). The adults represented in the crypt may not have lived in the parish or buried their children there, as there was a preference for people to be buried in the parish of their birth. Hence, the ratio of non-adults to adults in the crypt appears to be artificially low and not representative of fertility for the area at that time (Cox, 1996).

The sample represents individuals interred from August 1729 to 1859. They would have been relatively wealthy for most of the period under study, and many of the inhabitants were either independently wealthy or engaged in the silk weaving industry of East London (Molleson and Cox, 1993). The excavations in 1984 recovered 968 burials, 387 of known name, date of death, age and occupation. Of the total sample, 753 (78%) were adults and 215 (22%) non-adults (16 years and under). The number of preterm individuals was not stated (*ibid.*, 1993). The current study consisted of 189 non-adults and 17 preterm individuals, leaving nine unaccounted for. It is possible that some of these were among those cremated at the end of the excavation (Humphreys, *pers. comm.*). The preservation of the remains varied from the survival of garments and soft tissue to non-identifiable crystalline fragments. The coffins were packed into the crypt area and, as a result, some of the skeletons were damaged during their original interment and secondary deposition.

An extensive study of the known-age individuals in isolation was not within the scope of this study.

5.6. Representation of the Samples

When using data derived from cemetery samples it is important to remember that we are actually measuring *burial rates* and not *mortality rates*. Cultural practices may dictate if and where certain individuals were placed within a cemetery. Non-adults are often found to cluster and if the whole area of the cemetery is not excavated this could lead to an under-representation of the youngest members of the society. In some cases, the lack of non-adults in many Anglo-Saxon cemeteries has led authors to suggest that infants were buried elsewhere in the settlement (Sherlock and Welch, 1992; Harman *et al.*, 1981). Evidence for separate burial sites for infants in Britain is lacking but special cemeteries for infants have been discovered in Early Medieval Italy (Becker, 1995). During the Medieval Period, unbaptised and illegitimate infants were omitted from the cemetery (Daniell, 1997). Later, in the eighteenth and early nineteenth centuries, the practice of using wet-nurses meant that children from towns and cities, who died whilst being nursed, were often buried in the

countryside rather than being returned home (Fildes, 1988). This type of infant migration should be taken into account when attempting to assess mortality levels.

It was likely that, due to cultural factors, preservation problems and varied excavation techniques, the samples in this study would be disproportionate in their numbers of infants to older children, and non-adults to adults, compared to modern standards. In order to compare infant and child mortality rates between the sites, it was necessary to ascertain whether the samples were biased to the same degree. In addition, the different types of bias may provide extra information on the cultural factors influencing the samples.

Weiss (1973) estimated that 30-70% of a modern population would die before they reach 15 years of age. Therefore, if the number of non-adults under 17 years was representative of the sample, then the proportion of non-adults to adults should fall within this range. There was a noticeable difference between the proportion of non-adults in the urban and rural samples. At the rural sites of Raunds Furnells and Wharram Percy, non-adults make up 49% and 46% of the final sample respectively falling within the expected range proposed by Weiss (1973). In the urban groups, however, only 25% of the St. Helen and 21% of the Spitalfields sample were non-adults (Figure 5.7).

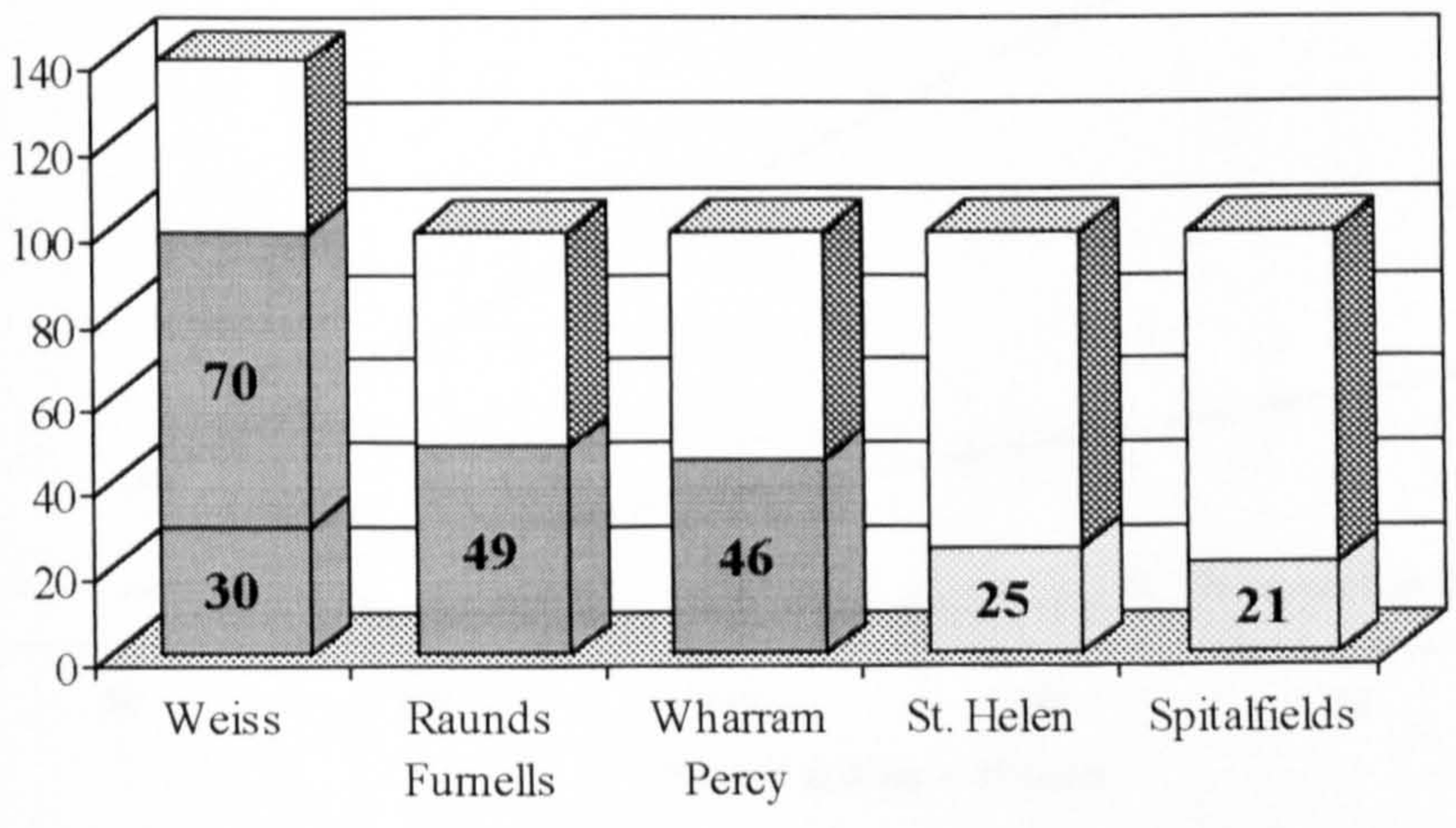


Figure 5.7 percentage of non-adults recovered from each site compared to data compiled by Weiss (1973).

In order to establish the extent of this shortfall, the proportion of infants to non-adults was also tested. Saunders and colleagues (1995) suggest that the number of neonatal deaths (birth to 27 days) should outweigh the number of post-neonatal deaths (28-364 days). However, this type of accuracy in ageing is difficult to achieve in archaeological samples. Alternatively, Brothwell (1971) suggests a 'rough' guide, which estimates the proportion of

individuals under one year of age, compared to all those under 20 years. Brothwell calculated that the 'normal' modern ratio for these groups is between 4:3 and 4:1. This procedure was applied to the data. Individuals were aged up to 49 weeks using regression equations of diaphyseal lengths provided by Scheuer and co-workers (1980), and plotted against the total number of individuals up to 17 years of age (Figure 5.8).

For all the sites, the samples proved to be biased, falling under the 4:1 ratio limit. Although these ratios would be slightly different when modified for individuals under 18 years of age, rather than under 20 years, it could not account for this shortfall. Therefore, although the proportions of non-adults to adults appear to be very different between the urban and rural sites, this bias appears to be similar for all the sites (Figure 5.8).

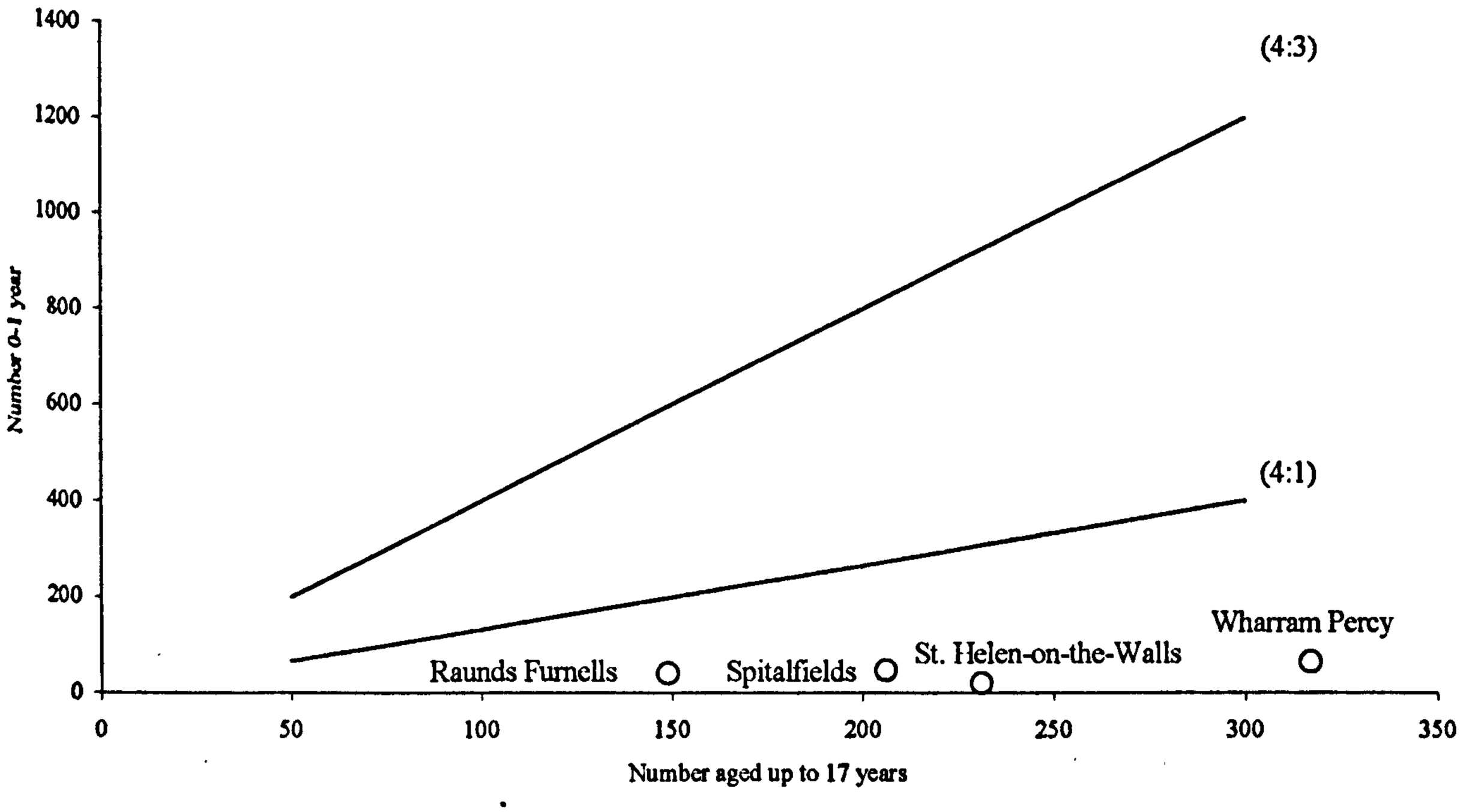


Figure 5.8. Proportion of infants to those dying up to 17 years of age.

A further test was carried out to qualify the differences in non-adult proportions. Buikstra and Mielke (1985) suggest that, in a representative group of non-adults, the probability of dying at birth, q(x), should be greater than dying at 15 years of age, and that at the age of 15, the probability of dying should be less than at 10 years (Figure 5.9).

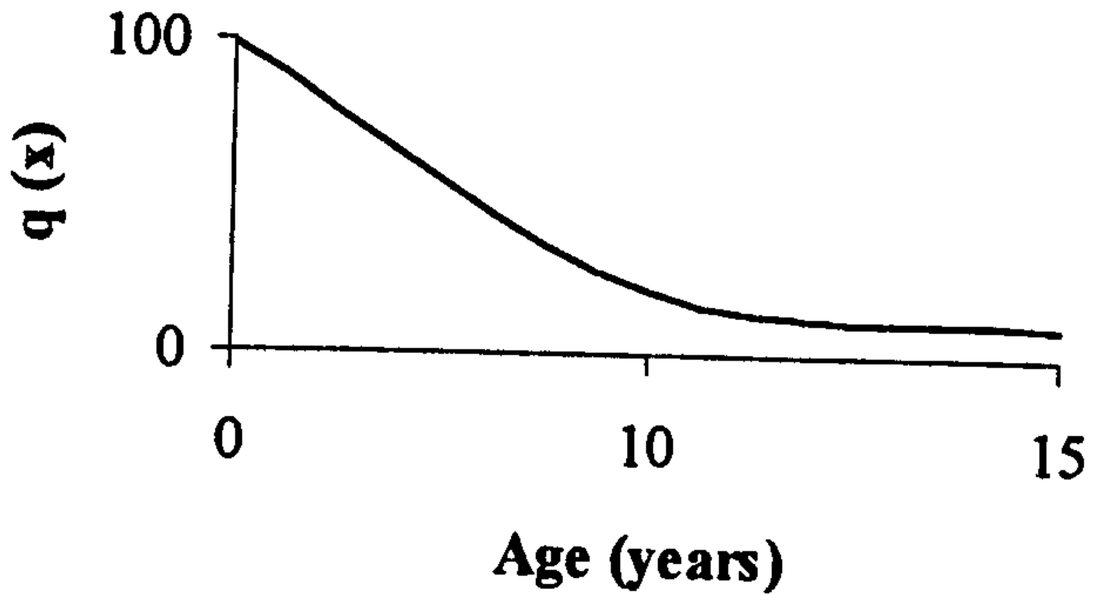


Figure 5.9. Expected pattern for probability of death at birth, 10 and 15 years.

In order to fit the data, the skeletons were placed into new age categories (0.0-0.99; 1.0-4.99; 5.0-9.99; 10.0-14.99; 15.0-17.0). The probability of dying at birth was greater than at 15 years of age for all except at St. Helen-on-the-Walls (Table 5.1). However, the probability of dying at 10 years was less than at 15 years suggesting that there was an excess of 10-15 year olds in the samples. This may be due to the preferential preservation and excavation of these larger non-adult remains, but may also be reflecting cultural factors. It is clear that all the samples are biased to some degree but, the amount of bias cannot be quantified or compensated for (Margerison, 1997).

Table 5.1. Probability of dying at birth, 10 and 15 years at study sites.

	q(x)	*Raunds Furnells*	*St. Helen*	*Wharram Percy*	*Spitalfields*
q_0	0.24		0.07	0.23	0.39
q_{15}	0.15 (0.76)*		0.17 (0.87)	0.15 (0.75)	0.14 (0.69)
q_{10}	0.62		0.76	0.76	0.61

*the numbers in brackets have been divided by five for comparison with q_0 (Margerison, 1997).

At St. Helen-on-the-Walls, it is already known that only two thirds of the cemetery was excavated. This is a common problem with urban excavations, where modern buildings artificially limit the extent of the cemetery. Nevertheless, the paucity of non-adults within St. Helen-on-the-Walls may reflect the actual nature of this population. A migratory population, such as that of York, may contain numerous young adults (perhaps 10-15 year olds) who delay marriage, and hence childbirth, until a later date, keeping fertility rates low (Goldberg, 1986). However, this would not account for the striking under-enumeration of infants (*n*=12).

In the crypt of Spitalfields, non-adults may have been omitted due to the cost of crypt burials and possible infant migration to the countryside. In addition, more adults than were contributing to birth rates may have been included in the crypt, due to the practice of people wishing to be buried in the parish of their birth.

5.7 SUMMARY

The four sites under study provide large non-adult sample sizes spanning AD 850-1859, and provide data for the health of non-adults in agricultural, urban and industrialised communities. The study of the relatively poor children from St. Helen and more wealthy children from Spitalfields also provides the opportunity to assess the impact of socio-economic status on environmental and cultural change. An analysis of the numbers of infants and children in each site seems to suggest that different cultural factors had an influence on the rural samples, and that, as these cemetery excavations are less likely to be limited by pre-existing structures, they have a better chance of being representative. However, all the samples had an under-representation of infants and an excess of 10-15 years olds.

CHAPTER SIX

METHODS

6.1 INTRODUCTION

The skeletons were divided into seven age categories: pre-term (<40 lunar weeks), newborn-0.5 years, 0.6-2.5 years, 2.6-6.5 years, 6.6-10.5 years, 10.6-14.5 years and 14.6-17.0 years. In the last age category, individuals were estimated to be older than 17 years when the root of the third molar was complete ('R_c': Moorrees *et al.*, 1963ab). By using large age categories it was hoped to reduce the errors introduced by inter- and intra-population variability (Lampl and Johnston, 1996).

In cases where no teeth were present, complete union of one or more of the following epiphyses was used to eliminate older individuals: the proximal humerus (14-20 years), distal radius (16-20 years), femoral head (13-18 years) distal femur (14-19 years) and/or proximal tibia (15-19 years) (Ubelaker, 1989: *75*). Owing to differences in maturation between the sexes, the latter method may have resulted in some older males being included, and some younger females being excluded from the study.

6.1.1 TERMINOLOGY

There are many different standards used for categorising non-adults. In this study, '*non-adults*' were individuals estimated to be 17 years or younger, '*infants*' were those under one year of age, '*children*' were aged between 1.0-14.5 years and '*adolescents*' between 14.6-17.0 years of age.

6.2 FOETAL AGEING

6.2.1 LONG BONE LENGTH

The use of diaphyseal lengths for estimating age at death is particularly useful in analysing foetal bone as the fragile developing dental crowns are often difficult to retrieve and are frequently lost during excavation and storage.

In 1978, Fazekas and Kósa carried out measurements of 138 Hungarian perinates of unknown age but known sex, who were either stillborn or had died shortly after birth. Ages were initially derived for the cadavers by comparing crown-heel length measurements to other modern studies. The individual bone elements were then measured and placed in age categories between 0.5-3.0 lunar months. Due to its relatively large sample size, these data have been used as a popular ageing method in biological anthropology. However, the exact time of fertilisation and actual age of the individuals was never known to the researchers (*ibid.*, 1978).

Owing to the problems with Fazekas and Kósa's ageing technique, it was decided to use the British standards developed by Scheuer and colleagues (1980), based on radiographs of perinates (from 24-40 weeks), using linear regression equations. Although the sample is more limited than the Fazekas and Kósa data (82 individuals) the actual age of the perinates was known. In the current study, pre-term individuals were classified as those under the age of 38-40 weeks gestation. All available bones were measured and the mean of the individual age estimates provided the final age.

6.2.2 Cranial Development

When the long bones were not available, the developing bones of the skull were used to provide an approximate age estimate (Figure 6.1).

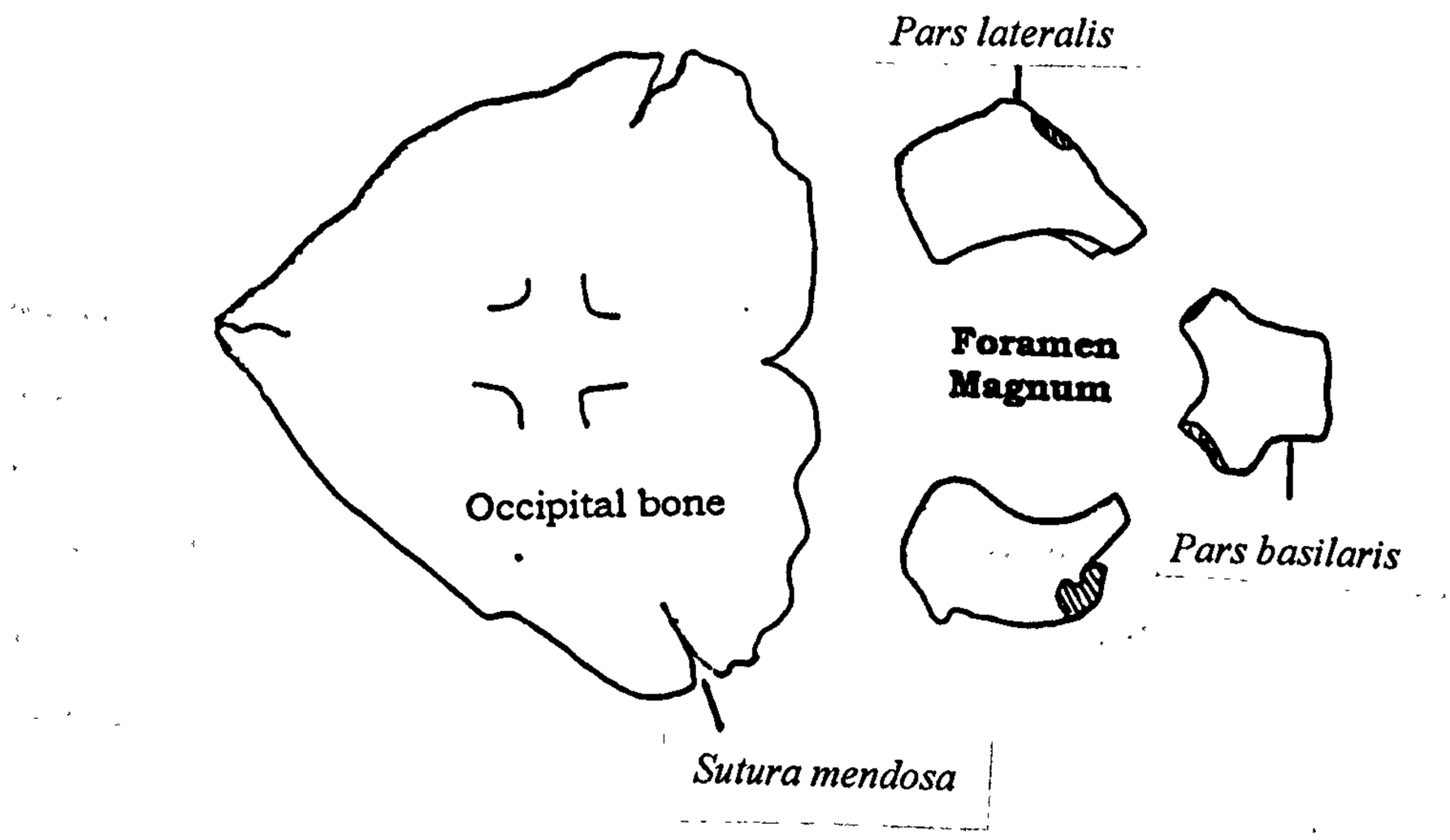


Figure 6.1. Schematic representation of the parts of the occipital bone. After Redfield, 1970:209.

6.2.2.1 Development of the *Pars Basilaris*

The *pars basilaris* was originally measured and described in detail by Redfield (1970) and later by Fazekas and Kósa (1978). Measurements of the maximum length (ML), sagittal length (SL) and maximum width (W) of the *pars basilaris* enabled a determination of the age of the foetus or infant (Figure 6.2). Redfield used different measurements to Fazekas and Kósa, but neglected to provide a diagram or description of the width measurement that he used. In 1994, Scheuer and MacLaughlin-Black tested this method on both archaeological and known aged individuals from nineteenth century England. They concluded that if the *pars basilaris* was longer than it was wide, the individual was less than 28 weeks *in utero*, and if the width was greater than the length, the individual was over five months of age. They suggested that the maximum width and sagittal length were of the greatest value when distinguishing between an early or late foetus, and the maximum width and maximum length were more accurate when identifying early or late infant material (Scheuer and MacLaughlin-Black1994). This method was used to determine whether the skeleton was pre-term or older than five months postpartum.

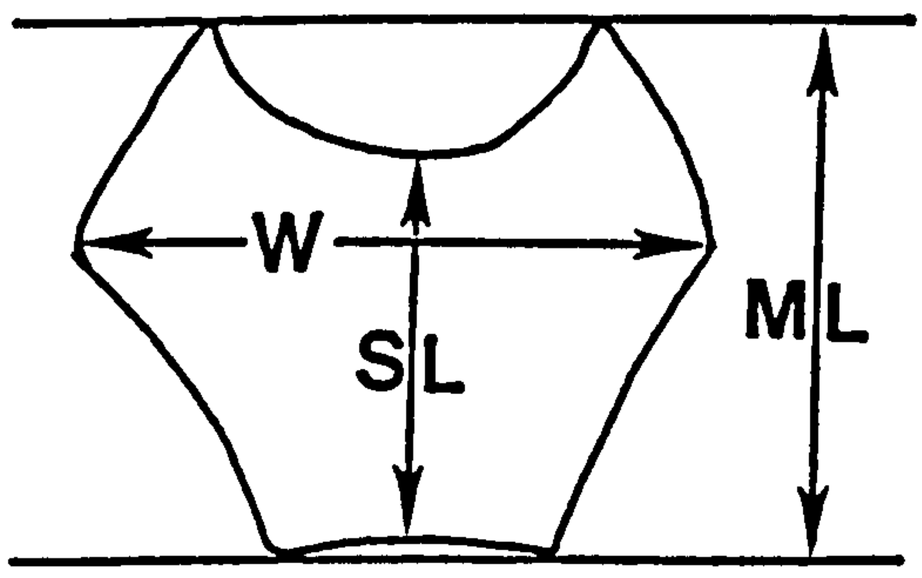


Figure 6.2. Measurements recorded from the pars basilaris. *From Scheuer and MacLaughlin-Black, 1994:*378.

6.2.2.2 The Occipital Bone

When preserved, the occipital bone was used to gain a rough estimate of age for the perinates. In his examination of 117 Yugoslavian skeletons and 19 German perinatal skulls, Redfield (1970) estimated that the *sutura mendosa* closed between one to four months, the *pars lateralis* fused to the occipital bone in 50% of individuals around the age of five years and, between the ages of six and 12 years, the *pars basiliaris* fused to the *partes laterales* (see Figure 6.1).

6.2.2.3 Development of the Temporal Bone

The development of the tympanic ring has also been widely used to distinguish between pre-natal and neonatal remains. Anderson first developed the method in 1960 and although he provided a good description and illustrations of the bone, he assigned no ages to the development other than 'foetal' and 'postnatal'. In 1978, Fazekas and Kósa studied the development of the tympanic annular ring in the foetal period and this method was extended in 1979, when Weaver recorded the development of the tympanic ring in the postnatal period using independent dental ages. In a following study, these criteria were tested on 300 known aged foetal and infant bones from the Hrdlicka collection (Curran and Weaver, 1982). Curran and Weaver found that there was a strong correlation between the developmental stages and chronological age, thus, when no dentitions were available, the tympanic ring provided an alternative method for estimating approximate ages for pre-term skeletons and those up to 2.5 years of age.

6.2.3 Perinatal Patterns of Mortality

Previous studies have used gestational ages at death of perinatal samples to assess evidence for infanticide (Smith and Kahila, 1992; Mays, 1993) and changes in mortality patterns (Saunders et al., 1995). Today, the majority of neonatal deaths are attributed to 'endogenous' factors, that is, physiological or organic weaknesses of the infant, whereas 'exogenous' factors are considered responsible for post-neonatal deaths (Landers, 1990; Saunders, 1992). Historical evidence has suggested that neonatal deaths, associated with endogenous factors are higher in rural areas than in urban communities, where environmental factors have a greater effect on child mortality, producing higher post-neonatal deaths (Vögele, 1994). Therefore, in order to assess whether the urban and rural samples had different patterns of infant mortality, individuals dying between the ages of 38-40 weeks of gestation and 41-49 weeks were compared. Gestational ages at death for pre-term (26-37 weeks) and infant (38-49 weeks) individuals were compared to data for stillbirths and infant deaths in Birmingham during 1947, derived from Gibson and McKeown (1951), and data for Romano-British burials obtained from Mays (1993), to test for any evidence of infanticide.

6.3 NON-ADULT AGE ASSESSMENT

6.3.1 INTRODUCTION

Age estimation of non-adult individuals is based on a physiological assessment of dental or skeletal maturation, and relies on its accurate conversion into chronological age. Error in the accuracy of this conversion can be introduced by random individual variation in maturation, the effects of the environment, disease factors, gender differences, secular changes and genetics (Demirjian, 1990; Saunders *et al.*, 1993). Variability in the timing of mineralisation and eruption of teeth between populations has been demonstrated in both the permanent and deciduous dentition by Tomkins (1996) and Holman and Jones (1998). Dental development is considered to be less affected by environmental influences than skeletal growth and maturation, and is the preferred method for producing an age estimate for non-adult individuals (Acheson, 1959; Schour and Massler, 1941).

In 1996, Lampl and Johnston warned that age at death estimates based on standards from modern healthy children, would result in considerable errors. When they tested the ages of similarly modern healthy children against the standards commonly used in biological anthropology, the children were under-aged by as much as four years for skeletal maturation and 3.5 years in their dental development. Therefore, greater errors should be expected when assigning ages to skeletal samples, due to the confounding environmental stresses suffered by the archaeological populations. In addition, variability between archaeological samples from different periods and environments may result in less than accurate comparisons for age-at-death and, hence, growth profiles (Lampl and Johnston, 1996). Using large age-at-death categories for comparative studies may help to reduce the degree of variability, and in this study it is precisely the variability introduced by environmental stresses that are of interest.

6.3.2 DENTAL DEVELOPMENT

The emergence of the primary and secondary dentition covers a period from the sixth month of postnatal life to around 20 years of age. The deciduous dentition forms between 12 -16 weeks prenatally to three years of age, when all the crowns are complete (Smith, 1991). The deciduous dentition is considered to be more robust than the permanent teeth to socio-economic conditions. However, Demirijian (1990) found that all the teeth, with the exception the first deciduous molar, emerged one month earlier in boys than in girls.

The development of the permanent dentition covers a wider time period than the deciduous teeth and forms from birth to around 12 years of age (Smith, 1991). Here, differences between sex are also evident with females being, on average, 1-6 months ahead in their development than males. This variability is also evident in individual teeth, and the canines are the most sexually dimorphic, with females often being as much as 11 months ahead of males in their development (Demirjian, 1990; Demirjian and Levesque, 1980). Environmental factors such as nutritional and socio-economic status have been shown to have a minimal impact on the timing of dental maturation (Garn *et al.*, 1973). However, caries or premature shedding of the deciduous teeth may delay the eruption of the succeeding permanent dentition (Demirjian, 1990).

The development of the deciduous and permanent dentition involves the calcification of the tooth crown and roots, resorption of the deciduous dental roots, and the subsequent eruption of the permanent teeth (Smith, 1991). Dental development follows a typical pattern and can be divided into convenient stages, beginning with cusp mineralisation and ending in root apex closure. In contrast, dental eruption is a continuous process with few biological markers and evidence of clinical eruption, or the emergence of the tooth through the gingiva, is distinct from the eruption though the alveolar bone seen in the hard tissues, which occurs slightly earlier (Hillson, 1992).

Dental age was determined using the standards derived by Moorrees and colleagues (1963a; 1963b) for the development and resorption of the deciduous dentition, and the development of the permanent teeth. This standard was based on longitudinal data from 246 white American school children, has small associated errors and provides a good sample size for each age category, spanning birth to 15 years of age. The method relies on observation of the deciduous canine, first and second deciduous molars, and six of the permanent mandibular teeth. A developmental stage is assigned to each tooth based on diagrams, and a mean age is read from a graph, with separate standards for males and females.

The graph is difficult to read and may have resulted in intra-observer error when each tooth was assessed. Hence, a table for the permanent dentition, derived from Moorrees and colleague's original chart and published by Smith (1991) was used, and the readings for males and females were averaged to take account of the unknown sex of the non-adults (Appendix I Table 1). Tables were also constructed for the development of the deciduous dentition, and male and female standards averaged (Appendix I Table 2). Although these data are based on a white North

American population who would be genetically similar to the white Europeans under study, different environmental conditions and nutritional status would have affected these children, and therefore some degree of error is expected.

The permanent canine was omitted from the analysis due to its greater variability in development between the sexes (Moorrees *et al.*, 1963b; Saunders *et al.*, 1993). Estimates were taken using either macroscopic assessments when the tooth could be removed, or using a radiograph. The final age was estimated by taking an average of the individual tooth ages. When the apex was complete (A_c) the tooth was omitted from the final age estimates, as it was not possible to estimate how long before death that stage of development had been reached. In some of the youngest individuals, the tiny developing crown had rotated in the jaw, making an accurate reading from the radiograph impossible. In addition, these developing crowns could not be visualised macroscopically without damaging the mandible. For this reason, an assessment of the dental development of the perinatal remains was not attempted.

6.3.2.1 Measurement of Error

There was some concern that the use of both macroscopic and radiographic methods to assign dental ages would result in inconsistencies, as it is easier to assign a higher stage of development when viewing the tooth itself. Radiographs will show a slightly later time for the formation of crowns and roots, than those based on dissected material, as the dental tissue needs a greater degree of calcification before being visible on a radiograph (Hillson, 1992). On a radiograph, teeth may rotate in the jaw and obscure the true developmental stage of the tooth. Similarly, fragile mineralised material, for instance the cleft and the start of root development, may be damaged resulting in a lower score for the macroscopic assessment.

Therefore, the developmental stages of the teeth from Spitalfields were assessed both radiographically and macroscopically. In 37 cases (71 teeth), the stages derived for teeth by both macroscopic and radiographic techniques were compared. There was conflict in the stages assigned to the teeth in 48% of the cases (34 teeth), with 21% (15 teeth) of the X-ray scores being lower than the macroscopic assessment, and 27% (19 teeth) of the X-ray scores being higher than the macroscopic assessment. In over half of the cases tested (52%; 37 teeth) the radiographic and macroscopic scores were in agreement and the mean difference between the ages assigned was 0.3 years.

Due to the large age categories employed in this study, in only four cases (9 teeth) the discrepancy between the stages assigned would have moved the individual from one age category to another. Nevertheless, comparisons of the growth profiles used one-year age cohorts and, therefore, a Friedman's two-way analysis of variance was carried out to test whether the differences between the scores, using these two methods, were significant. No significant differences were found at the 95% confidence level (X^2 =3.4, d.f.=1).

6.3.3 Long Bone Length

Although the dentition is the most accurate indicator of age in the non-adult skeleton, the teeth do not always survive and in these instances an age estimation can be made using diaphyseal lengths and the order and timing of epiphyseal union.

Maximum diaphyseal length measurements of the humerus, radius, ulna, femur and tibia were made using an osteometric board and recorded to the nearest millimetre. The left bone was chosen whenever possible but was replaced with the right when it was not preserved. Glued bones were only measured when a close union could be achieved. The standards provided by Ubelaker (1989) were then used to derive an age at death for those individuals with no dentition. Ubelaker studied the long bone lengths of the Arikara Protohistoric sample from Dakota USA which he initially aged using the dental calcification chart devised by Moorrees and co-workers (1963a,b). The measurements were then divided into one-year age categories up to the age of 18.5 years. As the skeletal development of a Native Indian population is slower than that of a Caucasian population, Ubelaker added a large standard deviation (±2 years) and a range of variation. This method was not ideal as in some cases there were no individuals to represent a particular age category (i.e. 8.5-9.5, 13.5-14.5 and 16.5-17.5). Others were constructed from data provided by only one individual. This method was considered most reliable for assigning an age to those between birth and 2.5 years of age, where larger sample sizes were available.

6.3.4 Bone Maturation

In the postnatal growth period, epiphyses begin to fuse when elongation of the diaphyseal shaft occurs at the expense of the epiphyseal cartilage, until it is completely replaced with bone. The last region of the growth plate to become completely ossified is usually at the bone margins and, therefore,

in dry bone, even though the majority of the plate has fused to the diaphysis, a groove may still be observed around the circumference (Roche, 1978); this is known as partial fusion. When the groove has completely disappeared, complete fusion is recorded. The ossified margin between the old epiphysis and diaphysis may still be visible on a radiograph for a number of years after the groove on the outer circumference has disappeared. Therefore, standards based on macroscopic assessment may record complete fusion at an earlier age than a radiographic study.

In this study the level of epiphyseal fusion was described as '*non-union*', '*partial union*' (when the line of union could still be seen macroscopically) or '*complete union*', when the fusion line was no longer visible to the naked eye (Buikstra and Ubelaker, 1994).

6.4 Life Tables

Life tables summarise a wide range of information on mortality, fertility, survivorship and life expectancy for a particular skeletal population and are derived from the original raw data of the number of individuals in each age category. Life tables based on skeletal populations assume a stationary population, where population size remains stable throughout the use of the cemetery, birth rates are regular and migration is non-existent or balanced (Boddington, 1984). Children represent the most demographically variable and sensitive section of the human life cycle (Roth, 1992). However, in palaeodemography they are frequently under-represented due to problems with interment, recovery and preservation and this has led some researchers to omit analysis of individuals under the age of 10 years in their studies (Buikstra and Konigsberg, 1985). However, Brothwell (1986-7) has suggested that the under-enumeration of infants in past populations may actually be a true reflection of the environmental stresses acting upon them. The expected pattern of high infant mortality is based upon modern demographic patterns where, before the 1930 s, postnatal mortality exceeded neonatal mortality in industrialised countries (Saunders *et al.*, 1995), and in developing countries deaths between one and four years of age are greater than infant deaths due to infectious disease (Brothwell, 1986-7).

In the previous chapter it was shown that individuals aged between birth and six months were under-represented in all the sites under study, with the urban samples having the lowest numbers of these individuals. Although this will affect the calculations of expectancy of life at birth, Moore and colleagues (1975) have suggested that infant under-representation only affects the

'survivorship' values, and will not affect the 'probability of dying' or 'life expectancy' values outside the infant age bracket. With this in mind, life tables were constructed in order to compare mortality rates between the sites, using the methods outlined in Boddington (1984) and Buikstra and Mielke (1985) where:

Age = osteological/dental age

D(x) = number in given age category

d(x) = number in given age category expressed as % of non-adults

l(x) = survivorship rate: **d(x)-l(x) from previous age category**

q(x) = probability of death at any given age interval: **d(x)/l(x)**

q2(x) = probability of death at any given age interval corrected for unequal age categories

L(x) = total number of years lived by individuals in each age category:

Years in age category (l(x)-lo/2: where lo = survivors in following age category

T(x) = total number of years remaining to be lived in each age category:

Sum of L(x)+L(x) values in following age categories

eox = expectation of life: **T(x)/L(x)**

For comparison, abridged life tables for non-adults dying in Bradford in 1856 and 1931 were created using data compiled by Milburn (1995) and based on records from Undercliffe Cemetery in the city of Bradford. Curves depicting mortality, probability of death (q2(x)), and age-specific life expectancy (eox) were produced. Differences between the mortality curves were tested using the Kolmogorov-Smirnov statistic (Siegel, 1956).

6.5 Growth Estimation

6.5.1 Intra-observer Error

In order to test the precision of the measurements taken by the observer, ten tibiae and ten femora from both Raunds Furnells and St. Helen-on-the-Walls were re-measured and the technical error of measurement (TEM) was calculated using the following equation:

$$TEM=\sqrt{\Sigma D^2/2N} \quad (6.1)$$

Where D is the difference between measurements, and N is the number of bones measured (Ulijaszek, 1998).

The coefficient of reliability (R) was then estimated to assess the percentage of variance between measurements created by measurement error, using the equation:

$$R=1-\{(TEM^2)/(SD^2)\} \quad (6.2)$$

Where SD is the total measurement error (Ulijaszek, 1998).

At St. Helen-on-the-Walls, the TEM was 0.43 cm and the coefficient of reliability was 0.97. At Raunds Furnells, the TEM was 0.34 cm and the coefficient of reliability was 0.98. This means that, in both cases, 90% of the variance in the measurements was due to factors other than measurement error. Raunds Furnells and St.Helen were tested due to ease of access but it is expected that the error of measurement would be similar in Wharram Percy and at Spitalfields.

6.5.2 Growth Profiles

Mean diaphyseal lengths of the long bones from individuals independently aged by their dental development were plotted against the dental age estimations (mean age) to produce skeletal growth profiles. Growth curves were plotted in one-year age cohorts in order to balance the mean ages in each age cohort. However this method reduces sample sizes. Gaps in the data were filled by averaging the measurements from the age above and below the missing value to provided a gradual curve in the data that would not interfere with its final interpretation.

Comparative data for the growth profiles were provided by the smoothed 50th percentile values published by Maresh (1955) from 1600 radiographs of healthy girls and boys, taking part in a longitudinal growth study by the Denver Child Research Council. Maresh published data for both males and females, and therefore the average of these measurements was used to take account of the unknown sex of the non-adults. Data was provided for females up to the age of 16.0 years and

for males up to 18.0 years. Between the ages of 10.0 and 12.0 years, Maresh provided data for both fused and unfused diaphyses. These measurements were used to calculate the percentage contribution of the epiphyses to the overall length of the bone for males and females. This percentage was then subtracted from the lengths given by Maresh for fused long bones between 12.6 and 17.0 years of age. This was necessary as only the diaphyseal length of the bones had been taken for the archaeological assemblage.

6.6 Indicators of Non-specific Stress

The frequency of cribra orbitalia, porotic hyperostosis, dental enamel hypoplasias and Harris lines were recorded in addition to indicators of non-specific infection: chronic maxillary sinusitis, new bone formation and endocranial lesions. The severity of the lesions, extent of healing, association between the lesions and the mean age at death for non-adults with and without stress indicators was investigated.

6.6.1 Indicators of Stress and Mortality

As nutritional stress and infection has been shown to have an effect on mortality, separate life tables were constructed for those with and without indicators of stress and disease in order to assess differential mortality patterns using the Kolmogorov-Smirnov statistic. When assessing the mean ages at death for those with and without hypoplasias, those without complete crowns (under 4 years of age) were omitted from the sample.

6.6.2 Indicators of Stress and Growth

Eveleth and Tanner (1990) have reported that the legs are the most sensitive to environmental stress as the femur and tibia are some of the fastest growing bones of the body. Therefore, femoral diaphyseal length measurements were chosen to assess the impact of stress on growth as the femora were better preserved than the tibiae in the combined sample (tibiae: n=246; femora: n=265).

6.6.3 CRIBRA ORBITALIA

Lesions indicative of cribra orbitalia are usually bilateral and range in severity from small isolated pits to the more severe 'hair-on-end' appearance. The lesions were graded according to the scheme described by Stuart-Macadam (1991: *109*) who provided photographs and a written description of the different appearances (see Appendix II) where:

0 - normal bone surface
1 - capillary-like impressions on the bone
2 - scattered fine foramen
3 - large and small isolated foramina
4 - foramina have linked into a trabecular structure
5 - outgrowth in trabecular form, from the outer table surface

An assessment of active and remodelled lesions was attempted in this study, using the descriptions provided by Mensforth and colleagues (1978). A lesion was considered '*active*' when the margins of the foramina were sharp, and '*healed*' when the foramina were filled in and the edges rounded; a combination of both healed and active lesions was also recorded. Porotic hyperostosis, a similar lesion on the parietals, was also recorded when present using the same method. Cribra orbitalia and porotic hyperostosis was considered '*absent*' when the majority of the orbital or cranial surface showed no evidence of pitting.

6.6.5 DENTAL ENAMEL HYPOPLASIAS

Dental enamel hypoplasias were recorded as furrows or pits on the dental enamel surface of the deciduous and permanent dentition. Measurements of the hypoplasias were taken from the occlusal margin of the defect, representing the beginning of the disruption, to the cemento-enamel junction (CEJ) on the anterior aspect of the crown. The measurements were taken with Mityoto digital calipers that had a precision of 0.01mm and an accuracy of ±0.02mm. To avoid over-recording these features, well-defined defects were measured when a dental probe could be placed into the groove, and the line followed around the tooth. In order to be sure of recording non-specific hypoplasias, rather than those caused by localised trauma or infection (Turner teeth), more than two teeth, on opposite sides of the jaw, had to be affected before 'presence' was recorded (Goodman and Rose, 1990; Schultz, 1992; Skinner, 1986). Hypoplasias were only considered absent when four or more anterior teeth were available for examination and did not display a defect.

The age of the individual at the time of defect formation was calculated using the following equation:

$$X-\{(YF/CH) \times DH)\} \qquad (6.3)$$

Where X is the age of the individual at the time of crown completion (using Moorrees et al.*, 1963a,b), CH is the mean crown height, and DH the distance between the defect and the cemento-enamel junction (Figure 6.3) (Goodman and Rose, 1991).*

The mean crown height was taken from the unworn teeth from each sample in order to provide an average for that population, and to eliminate any variability in the development of the individual teeth. The permanent mandibular canines were selected for the analysis as they have, along with incisors, been found to display hypoplasias more clearly and frequently than any other tooth, with the exception of the maxillary central incisors (Condon, 1981; Goodman and Armelagos, 1985; Santos and Coimbra, 1999). In addition, the canine crowns develop until four years of age (Moorrees *et al*., 1963b) and therefore provide a record of postnatal stress from 0.5 to 4.0 years. Standards for dental development provided by Moorrees and colleagues were included in equation 6.3 in order to provide the 'best-fit' with the age at death calculated for the samples. The canines were used as they represented the best teeth for this method, despite the caveats discussed in section 6.3.2.

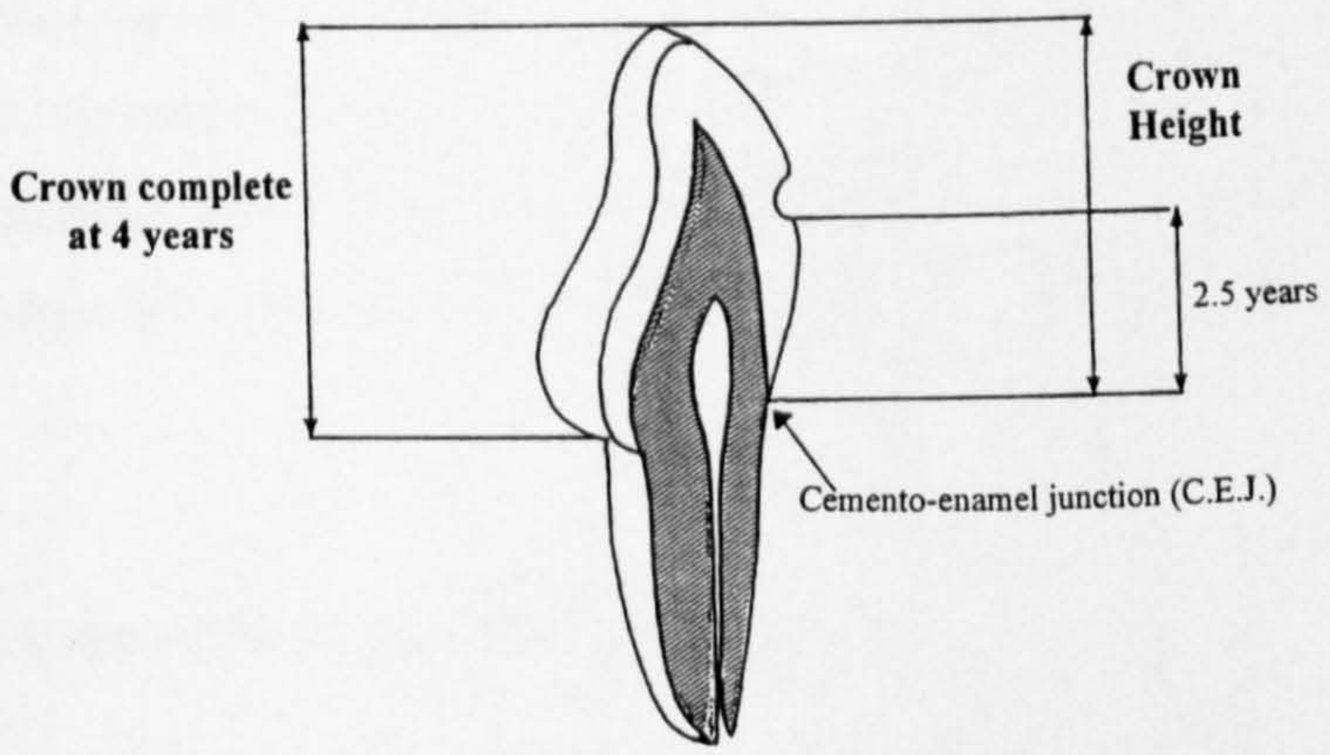


Figure 6.3 Location of measurements taken to derive age at defect formation on the canine crown.

Sciulli (1978) suggested that deciduous teeth are less likely to display defects due to stronger developmental canalisation and a more stable prenatal environment. However in skeletal material,

the prevalence of deciduous dental defects are likely to be underestimated as non-adults shed their primary dentition around 6-8 years of age. Hence, any prenatal stress will not be apparent in the older non-adults. In the younger skeletons, unerupted deciduous teeth cannot be recorded and will also result in an under scoring of prenatal defects.

6.6.5 Harris Lines

The present study provided an opportunity to estimate the prevalence of Harris lines in a large number of non-adults before the lines had remodelled. For St. Helen-on-the-Walls, radiographs were taken of all intact tibiae at 70 kV on a Hewlett-Packard Faxitron Series 43805 X-ray system and the exposure time was reduced from 2.0 to 0.8 seconds depending on the size and density of the bone. The tibiae from Raunds Furnells had already been X-rayed in a previous study (Ribot and Roberts, 1996) using the same machine and settings. To save costs, pre-existing radiographs were used whenever possible. At Wharram Percy, radiographs of the femora were provided by Simon Mays of English Heritage, but for the Spitalfields collection, only radiographs of the youngest individuals had been taken (up to 2.6-6.5 years) and, therefore, any analysis of Harris lines in the older age groups for this sample could not be undertaken.

Well-defined lines of increased radio-opacity were recorded when they extended more than half way across the distal aspect of the bone (Hummert and Van Gerven, 1985). The frequency and mean number of both faint lines (scored as '1') and strong lines (scored as '2') within the different age groups was calculated. Initially it was hoped to estimate the age of the individual at the time of Harris line formation (Maat, 1984). However, the use of radiographs produced by different machines and from different researchers, using different bone elements, meant that the error in these measurements would have been too great to make such estimations plausible.

6.7 Indicators of Non-specific Infection

6.7.1 Chronic Maxillary Sinusitis

In younger individuals the sinuses are not complete enough to determine the presence of any infection. Therefore, maxillary sinusitis was only recorded in the individuals from the 2.6-6.5 year age category onwards. Unfortunately, intact sinuses could not be examined and only non-adults with open sinuses were examined (*i.e.* damaged postmortem allowing visualisation).

Dental diseases such as caries, abscesses and oro-antral fistulae were recorded in an attempt to separate those with 'dental' induced sinusitis from those with sinusitis of another, perhaps respiratory, aetiology. Conditions such as nasal polyps, allergies and acute respiratory infections could not be directly identified.

Care was taken not to confuse broken root caps (thin bone caps covering a root protruding into the antrum) with smooth walled oro-antral fistulae, indicative of an abscess draining into the sinus cavity. In addition, the large growth pits, formed by the expanding sinus floor to accommodate the developing molars were not recorded. The following types of lesion were recorded (see Appendix III for illustrations) (Boocock *et al.*, 1995; Lewis *et al.*, 1995):

(a) White or grey deposits of woven bone
(b) Isolated spicules
(c) Clusters of connected spicules
(d) Remodelled plaques of lamellar bone

6.7.2 New Bone Formation

Eight grades of non-specific infection were recorded on the long bones. A lesion was considered absent when the tibiae and fibulae were present but none of the bones showed signs of infection, and was recorded as present if any of the long bones were affected. The exact position and distribution of the lesions was recorded graphically and the nature of the infection graded using the criteria outlined below (see Appendix IV for illustrations). The following grades do not necessarily correspond to the severity of the infection, but rather the state of healing and the extent to which the bone is affected:

(1) Inflammatory pitting
(2) Localised deposit of woven bone covering up to one third of the shaft
(3) Diffuse woven bone. Covering two thirds of the shaft.
(4) Plaque of lamellar bone
(5) Diffuse lamellar bone
(6) Mix of lamellar and woven bone (representing a re-occurring infection)
(7) Sclerosing osteomyelitis (osteitis)
(8) Osteomyelitis

6.7.3 ENDOCRANIAL LESIONS

Endocranial lesions were not recorded in the pre-term or 0-0.5 year age categories due to the problem of distinguishing 'growth' deposits of primary bone from indicators of trauma or infection in the youngest skulls. Various forms of the lesion have been identified on the endocranial surface (see Brothwell in Lilley *et al.*, 1994: 465-466) and may represent different aetiologies (Illustrations in Appendix VI). Here the distribution of the lesions was recorded and the types described as:

(a) New bone formation
(b) Capillary-like formations, new bone expands around vascular structures
(c) Remodelling deposits of white or grey woven bone
(d) 'Hair-on-end' trabeculae.

6.8 METABOLIC DISEASE, DENTAL DISEASE AND OTHER CONDITIONS

Evidence for rickets, scurvy (see Chapter Four), trauma, dental disease (Hillson, 1996) and other pathological conditions were recorded in an attempt to determine whether the prevalence of these conditions was different in the contrasting environments and between time periods.

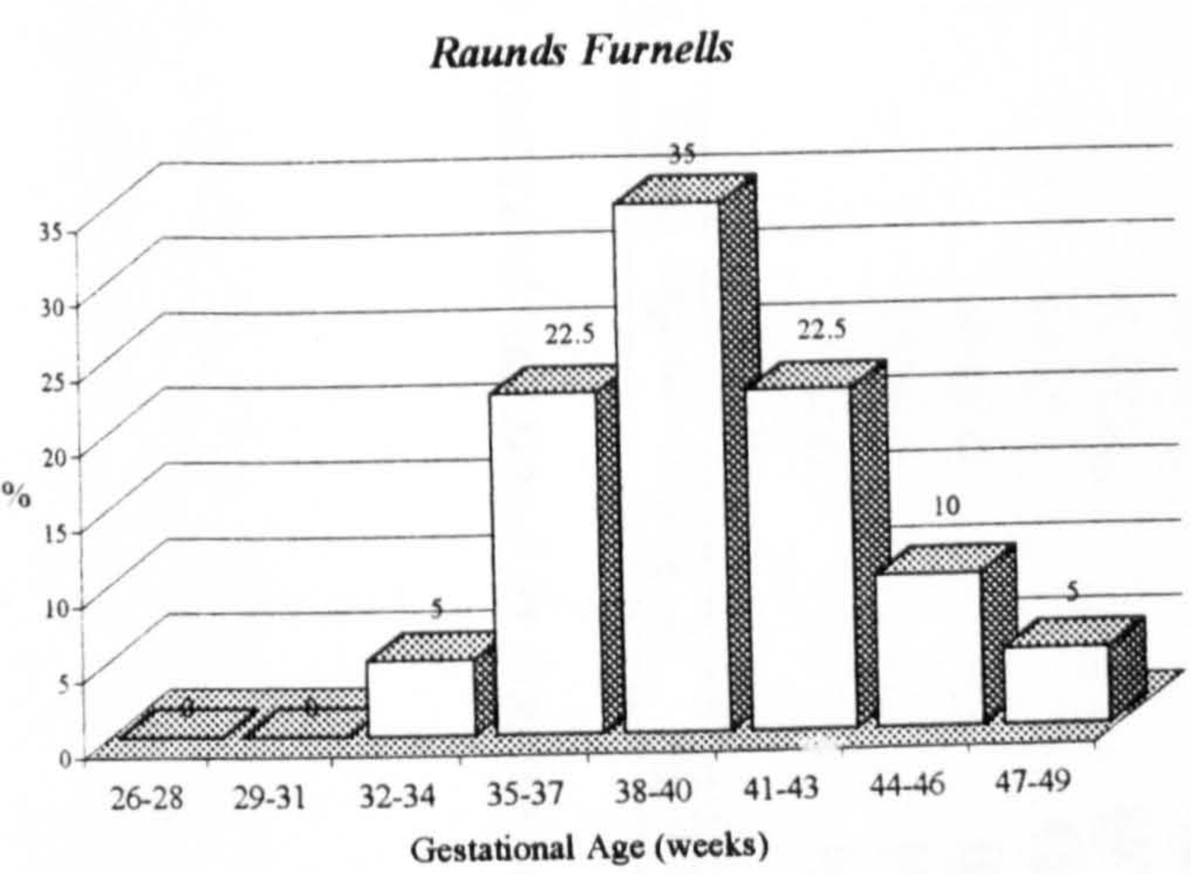
CHAPTER SEVEN

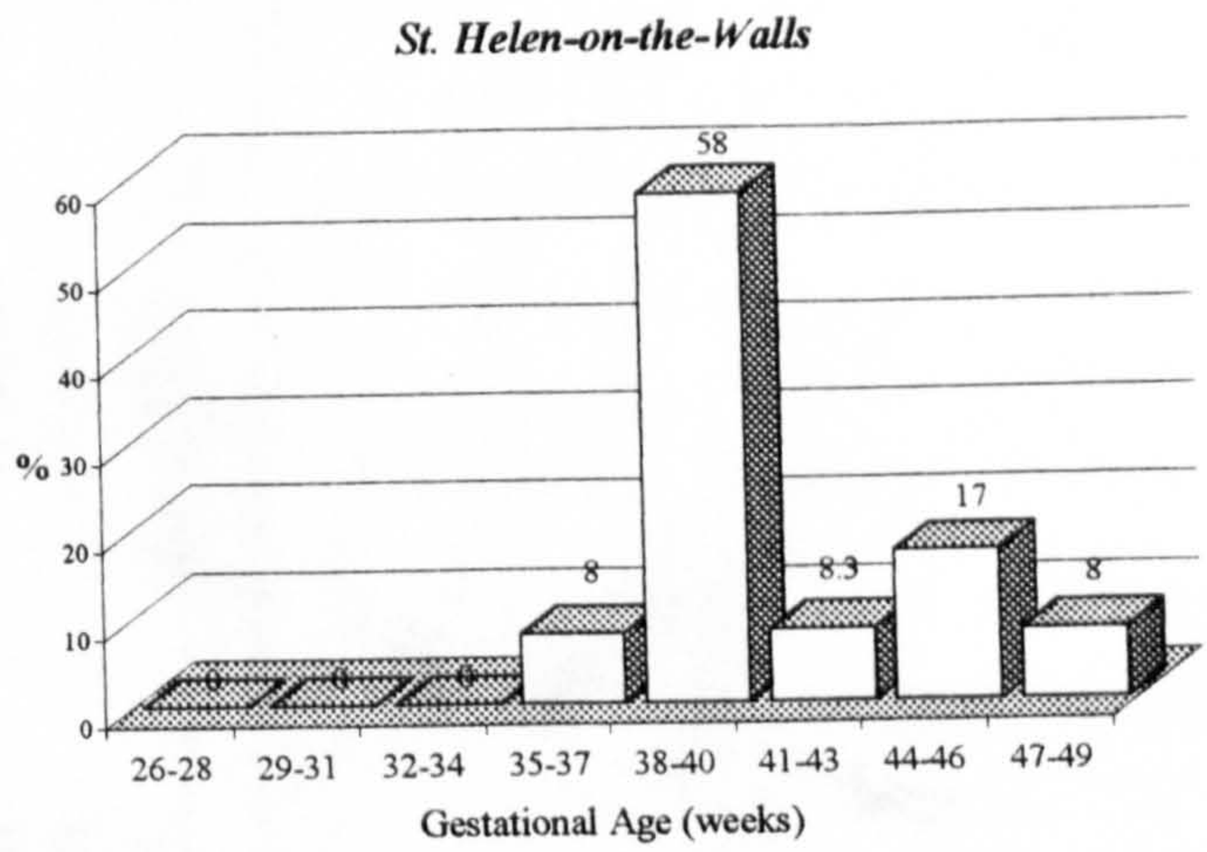
RESULTS

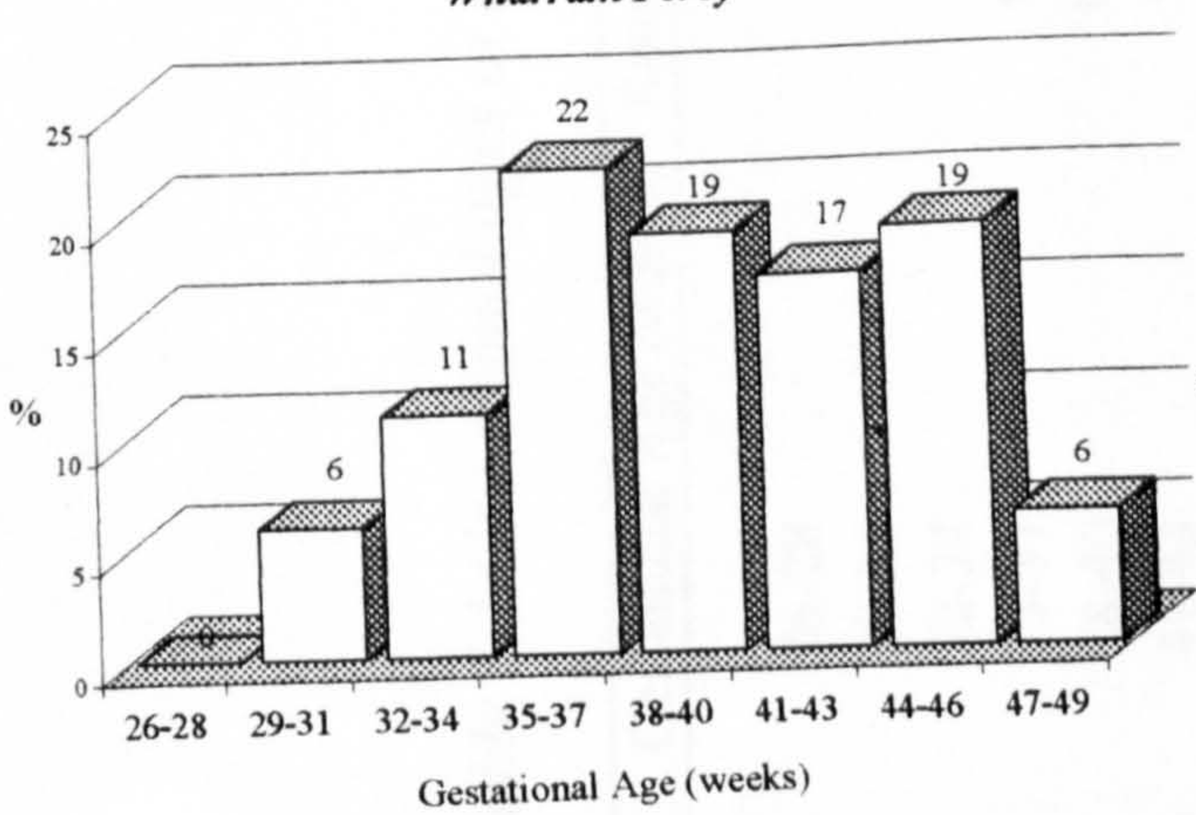
7.1 INFANT MORTALITY

7.1.1 PERINATAL DEATHS

The ages of individuals between 26 and 49 weeks were estimated using the method of Scheuer *et al.* (1980) (Figure 7.1; Table 7.1). Perinatal deaths can occur as the result of poor maternal health, lack of foetal integrity, during childbirth, as the result of exogenous environmental factors or, due to infanticide. Archaeologically a foetus is considered developed enough to be viable between 38-40 weeks gestation (Saunders, 1992) and, for the purposes of this study, this period was taken as the time of birth. Any individuals below this age were considered to have been spontaneously aborted, stillborn or premature.







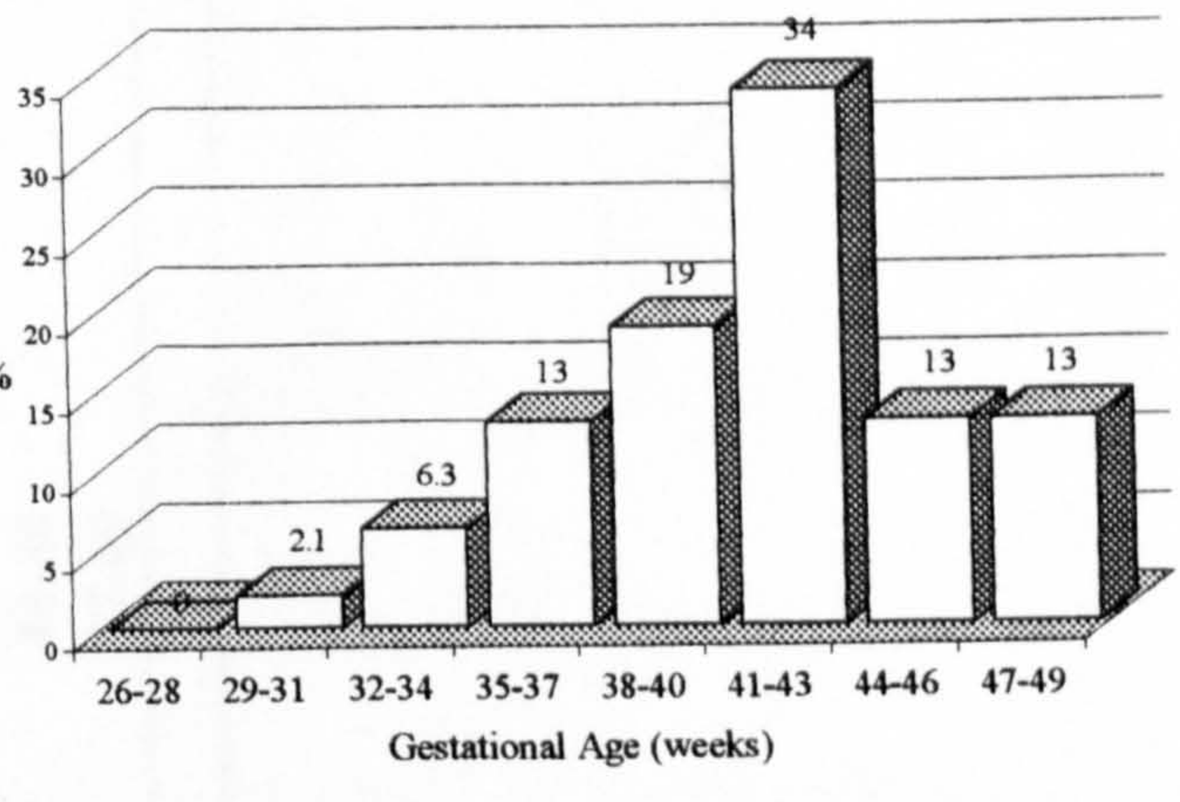


Figure 7.1. Perinatal Mortality (RF = 40 individuals; SH =12 individuals, WP = 64 individuals; CS = 47 individuals)

Table 7.1 Gestational ages at death (weeks) of preterm, perinatal and infant individuals.

Gestational Age (weeks)	Raunds Furnells	St. Helen-on-the-Walls	Wharram Percy	Christ Church Spitalfields
	n (%)	*n (%)*	*n (%)*	*n (%)*
26-28	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
29-31	0 (0.0)	0 (0.0)	4 (6.3)	1 (2.1)
32-34	2 (5.0)	0 (0.0)	7 (11.0)	3 (6.4)
35-37	9 (22.5)	1 (8.3)	14 (21.8)	6 (12.8)
38-40	14 (35.0)	7 (58.3)	12 (18.7)	9 (19.1)
41-43	9 (22.5)	1 (8.3)	11 (17.2)	16 (34.0)
44-46	4 (10.0)	2 (17.0)	12 (18.7)	6 (12.8)
47-49	2 (5.0)	1 (8.3)	4 (6.3)	6 (12.8)
	40	12	64	47

At Raunds Furnells and St. Helen, the majority of perinatal deaths occurred around the time of birth (35% and 58% respectively). At Wharram Percy however, the deaths were more evenly distributed with the majority of deaths occurring between 35-37 weeks (22%). At Spitalfields, the highest death rates were later than the other sites, and occurred in the 41-43 week period (34%).

The Kolmogorov-Smirnov statistic (Siegel, 1956) indicated that none of the overall distributions differed significantly form each other at the 95% confidence level. However, the chi-squared statistic, applied to numbers of individuals (Kirkwood, 1988), revealed that there were significantly more infants dying in the 38-40 week age category at St. Helen than in the same age category at Spitalfields ($X^2 = 6.99$, P = 0.01) or Wharram Percy ($X^2 = 7.59$, P = 0.01). This peak in deaths during the birth period occurred despite a paucity of data for perinates at St. Helen (n=12).

7.1.2 Neonatal versus Postnatal Mortality

In order to assess whether predominantly endogenous or exogenous factors were responsible for the infant deaths, and whether these patterns were different for the urban and rural sites, percentages of neonatal deaths (26-40 weeks) and post-neonatal deaths (41-47 weeks) were compared (Figure 7.2).

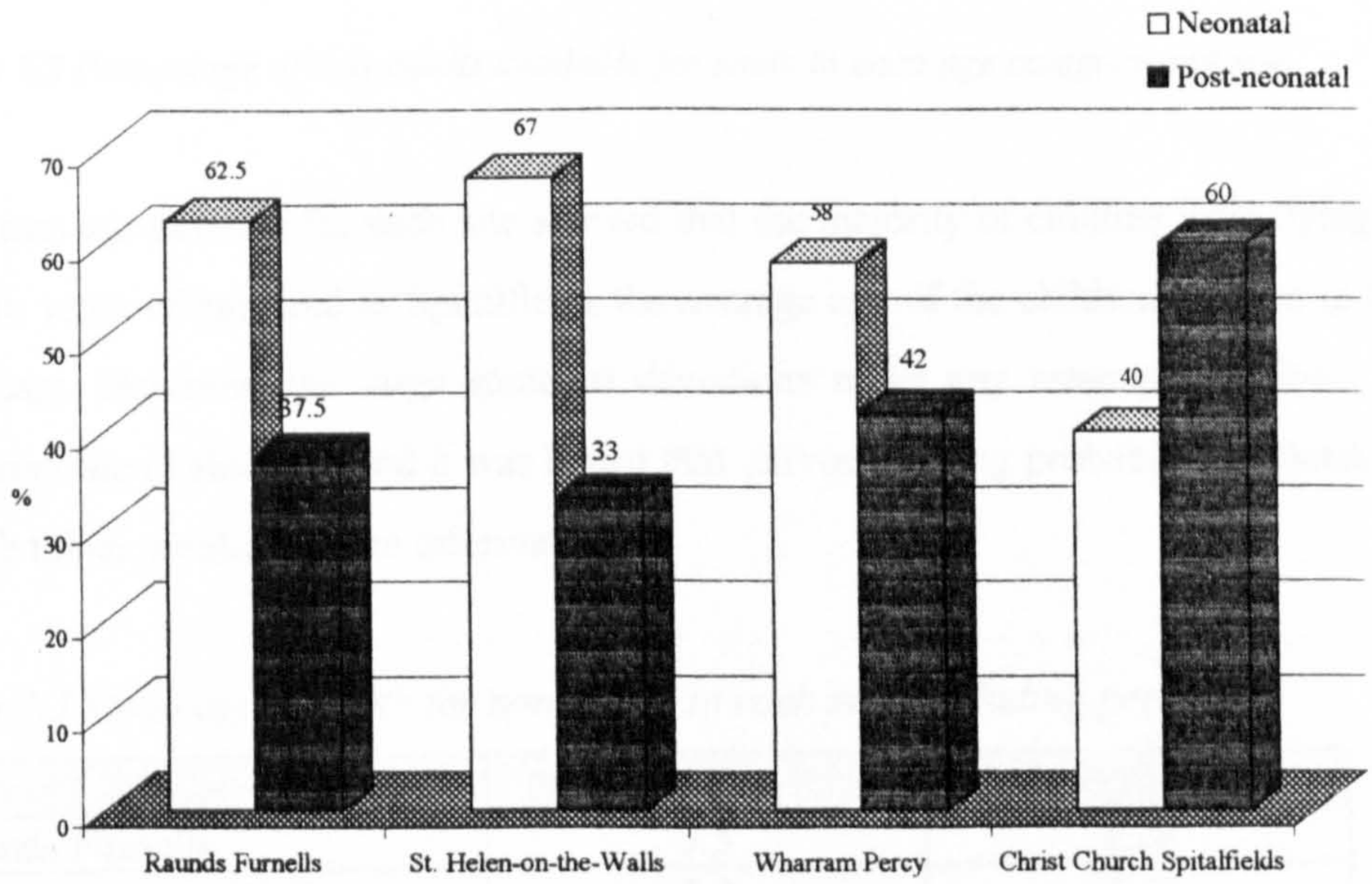


Figure 7.2 Neonatal versus post-neonatal mortality

In all the sites, with the exception of Spitalfields, neonatal mortality, indicative of endogenous mortality factors, outweighed post-neonatal mortality.

7.2 Demographic Profile of the Non-adults

Table 7.2 and Figure 7.3 illustrate the number and percentage of skeletons available for study in each age category. In all the samples, some data was lost due to poor preservation that prevented the skeletons being assigned an age.

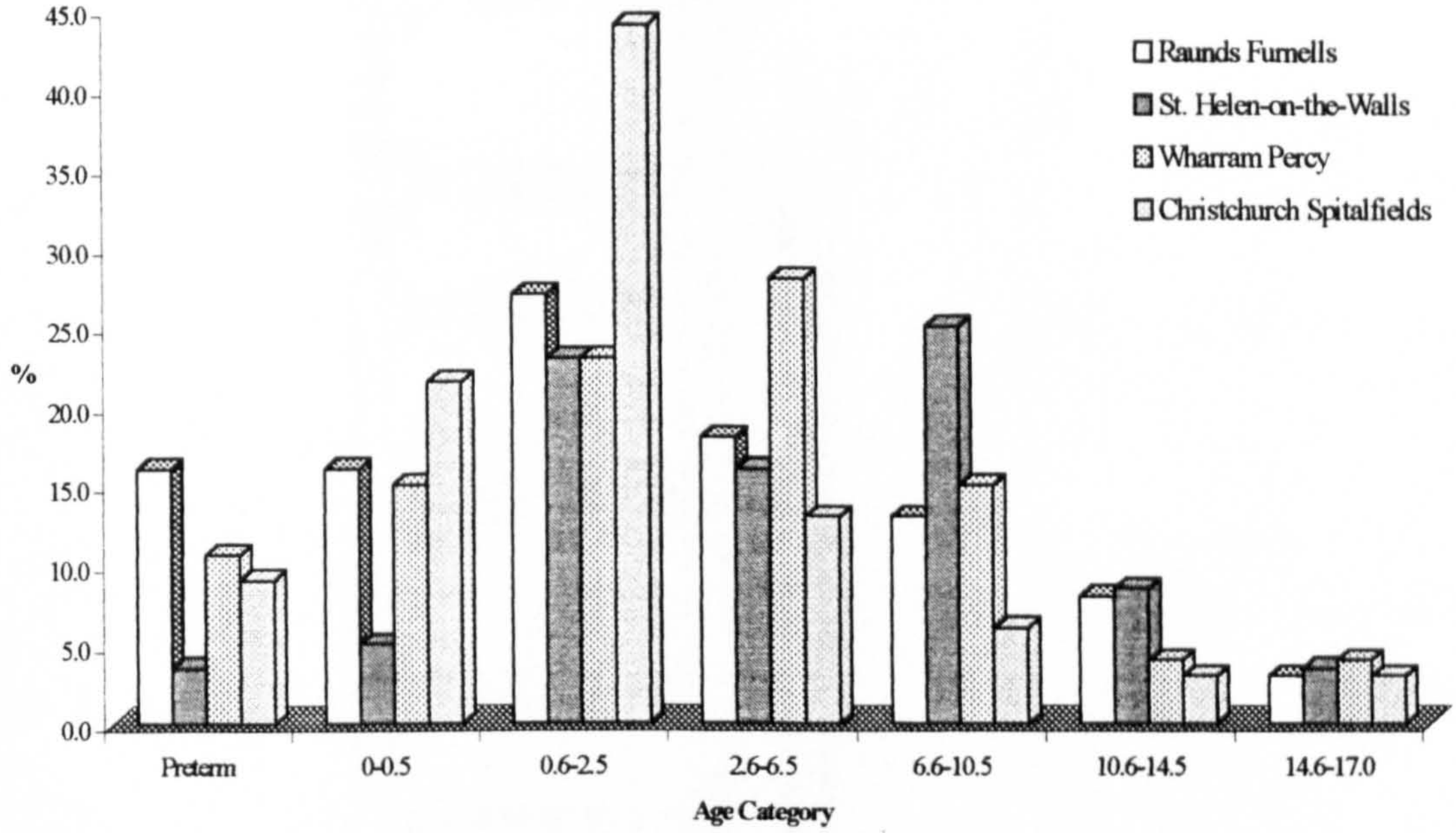


Figure 7.3 Percentage of non-adults available for study in each age category and site.

The mean age at death for each site showed that the majority of children were dying between five and six years of age, and at Spitalfields the average age of the children interred in the crypt was 2.9 years. However, the large standard deviations make any assumptions about these results inappropriate (Table 7.3) and it was hoped that curves showing probability of death derived from the life tables would be more informative.

Table 7.3 Mean age at death for non-adults in each site (excluding perinates)

Site	*Mean*	*sd*
Raunds Furnells	5.5	*4.26*
St. Helen-on-the-Walls	5.8	*3.55*
Wharram Percy	4.6	*3.98*
Christ Church Spitalfields	2.9	*3.38*

Table 7.2 Demographic profile of the non-adults

	Raunds Furnells			St.Helen-on-the-Walls			Wharram Percy			Christ Church Spitalfields		
Age	*Number*	*% Non-adults*	*% Total Population*	*Number*	*% Non-adults*	*% Total Population*	*Number*	*% Non-adults*	*% Total Population*	*Number*	*% Non-adults*	*% Total Population*
Preterm	23	16.0	6.0	7	3.5	0.7	32	10.6	4.6	17	9.0	2.0
0-0.5	23	16.0	6.0	10	5.0	0.9	46	15.0	15.0	40	21.5	4.0
0.6-2.5	38	27.0	10.0	45	23.0	4.0	70	23.0	10.0	82	44.0	8.0
2.6-6.5	25	18.0	7.0	64	16.0	6.0	85	28.0	12.5	24	13.0	2.0
6.6-10.5	18	13.0	5.0	49	25.0	5.0	46	15.0	7.0	12	6.0	1.0
10 6-14.5	11	8.0	3.0	18	8.5	2.0	13	4.0	2.0	6	3.0	0.6
14 6-17.0	4	3.0	1.0	7	3.5	1.0	11	4.0	2.0	5	3.0	0.5
Total	142 (of 363)		39.0	200 (of 1041)		19.0	303 (of 681)		19.0	186 (of 968)		19.0

Raunds Furnells: 7 (5% non-adults) could not be aged and were excluded from the study; St. Helen-on-the-Walls: 31 (15.5% non-adults) could not be aged and were excluded from the study; Wharram Percy: 16 (5% non-adults) could not be aged and were excluded from the study; Christ Church Spitalfields: 20 (10% non-adults) could not be aged and were excluded from the study.

7.3 LIFE TABLES

Life tables for the four study sites, and for non-adult deaths in Bradford in 1856 and in 1931 are presented in Appendix VI.

7.3.1 MORTALITY CURVES (d(*X*))

Mortality curves were constructed based on cumulative percentages, in order to statistically test the differences between the archaeological populations and to compare the archaeological with the historical data from Bradford. Due to the problems of under-enumeration (discussed in Chapter Five), any conclusion about the different mortality patterns of these samples was confined to the probability of death and expectancy of life profiles.

Figure 7.4 illustrates the mortality patterns for both the archaeological and historical samples. The later medieval sites of Wharram Percy and St. Helen followed a similar mortality pattern with the exception of the earliest category where they differed significantly ($KS_{0.05}$:0.12) due to the under-enumeration of infants at St. Helen. For both of these samples the peak in mortality occurred between the ages of 2.6 and 6.5 years.

Interestingly, Raunds Furnells and Spitalfields, although having very different mortality profiles in the 0.6-2.5 year age category ($KS_{0.5}$:0.16), followed a similar pattern. The later medieval sites differed significantly from industrial Spitalfield's mortality profile until the 10.6 year age category (SH $KS_{0.01}$:0.25; WP $KS_{0.001}$:0.22).

When the sites were compared to the historical data from Bradford, all the archaeological samples showed an under-enumeration of infants, resulting in an artificial later peak at the 0.6-2.5 year age category, rather than a steady decline after birth. The Bradford data also showed a dramatic decline in infant mortality between 1856 and 1931 which is consistent with historical trends (see Chapter Two).

7.3.2 PROBABILITY OF DYING (q2(*X*))

This statistic describes the probability that having reached a certain age category one will die within it. Curves based on the probability of death in each age category are more reliable than

those based on mortality curves (using d (x)) as they are not based on the number of individuals in each category and are therefore, least likely to be affected by under-enumeration after the earliest age category (Moore *et al.*, 1975). When compared to data derived from burial registers for Bradford in 1856 and 1931, the curves of the archaeological samples roughly followed a similar pattern to those seen in Bradford (Figure 7.5), with a high probability of death at birth, which declined as the children reached their sixth month of life and increased again at 17 years where the data ends.

At birth, the probability of death was highest at Spitalfields (0.46), but this still fell well below the values of the historical samples (Bradford 1856 = 0.84; 1931 = 1.18) and at St. Helen, the levels at birth were especially low (0.10) due to the severe under-representation of infants in this sample. Interestingly, in both later medieval sites, that is St. Helen and Wharram Percy, the number of children dying between 6.6-10.5 years significantly increased but this may be the result of small sample sizes in the other sites (RF=18; SH=49; WP=46; CS=12). At Wharram Percy, the probability of death drops dramatically during the next age category, and it is possible that some older individuals were under-aged and placed in the earlier 6.6-10.5 year age category. However probability of death levels remain high at St. Helen. The Bradford data showed that only seven children in 1856, and four children in 1931, died during this age category.

7.3.3 Expectation of Life (*EOX*)

Curves derived from the expectancy of life column in a life table (*eox*) refer to the number of years one might expect to live once a certain age has been reached. The rural sites of Raunds Furnells and Wharram Percy had similar expectancies of life at birth, with children expecting to live 4.3 and 4.4 years respectively (Figure 7.6). At St. Helen, the expectancy of life was higher with children living for 5.5 years after birth. However, the small number of individuals in this age category makes this figure artificially high. All of the medieval sites have life expectancies at birth much higher than the children from the industrial periods in London and Bradford. The expectancy of life at birth at Spitalfields (2.9 years) is higher than that of children born in Bradford in 1856 (1.7 years) and similar to those born in 1931 (2.7 years). The low life expectancy in 1856 is not surprising as Industrial Bradford had the worst infant mortality rates in the country during the nineteenth century (Thompson, 1984). Despite some discrepancies at birth, by the 2.6-year age category, the archaeological sites followed a similar pattern to the curves derived from the Bradford burial registers.

Figure 7.4 Mortality Curves

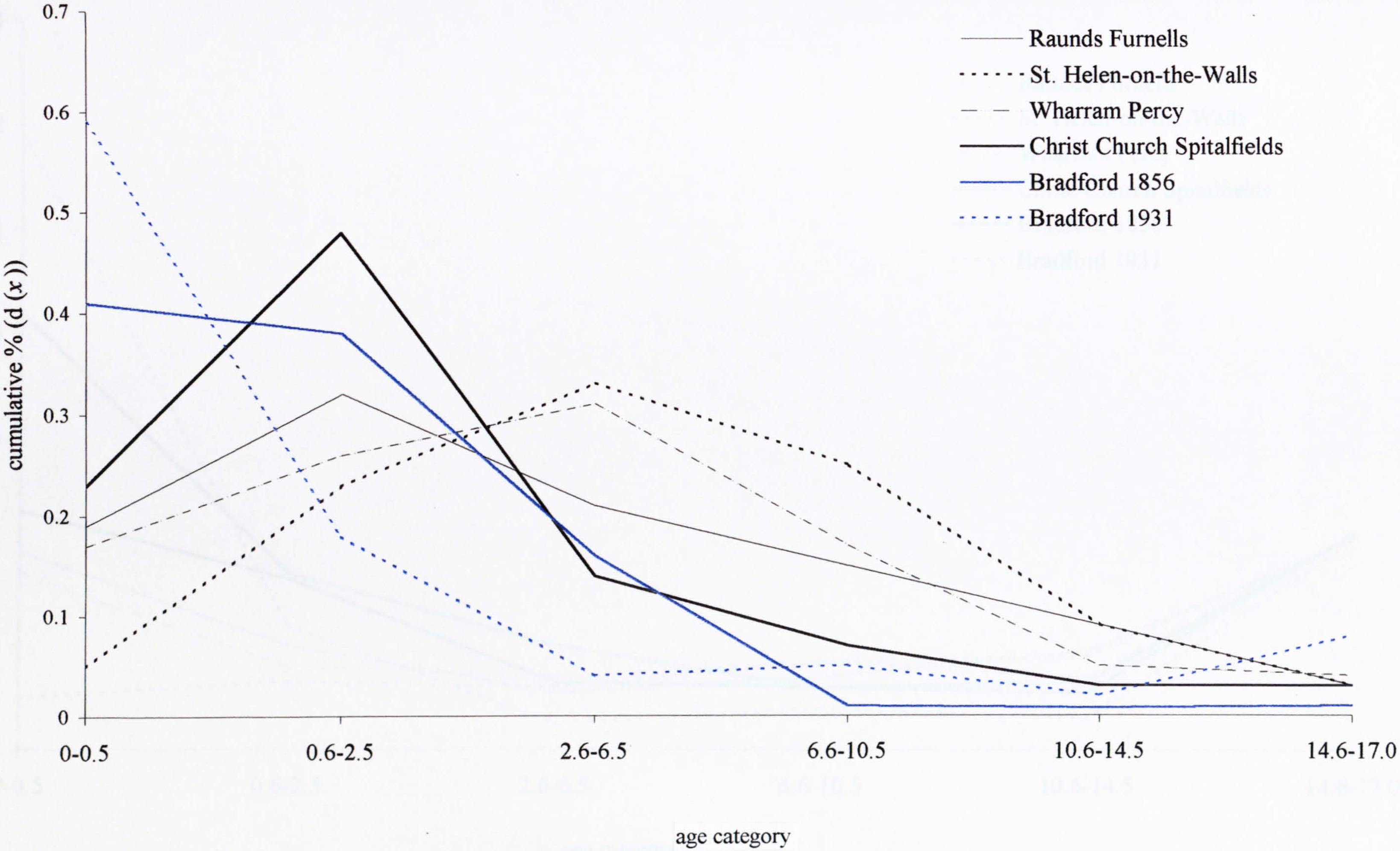


Figure 7.5 Probability of Death

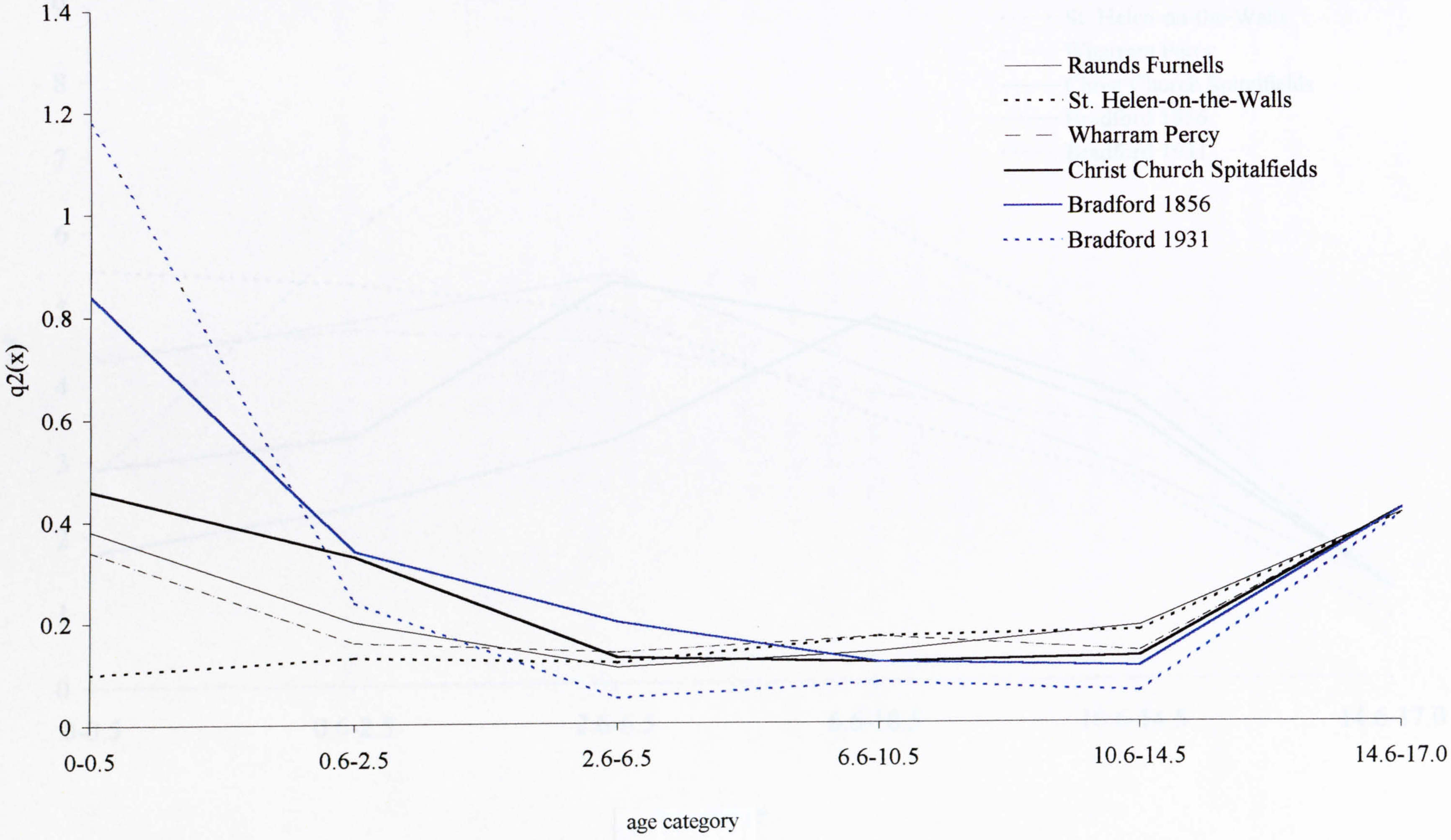
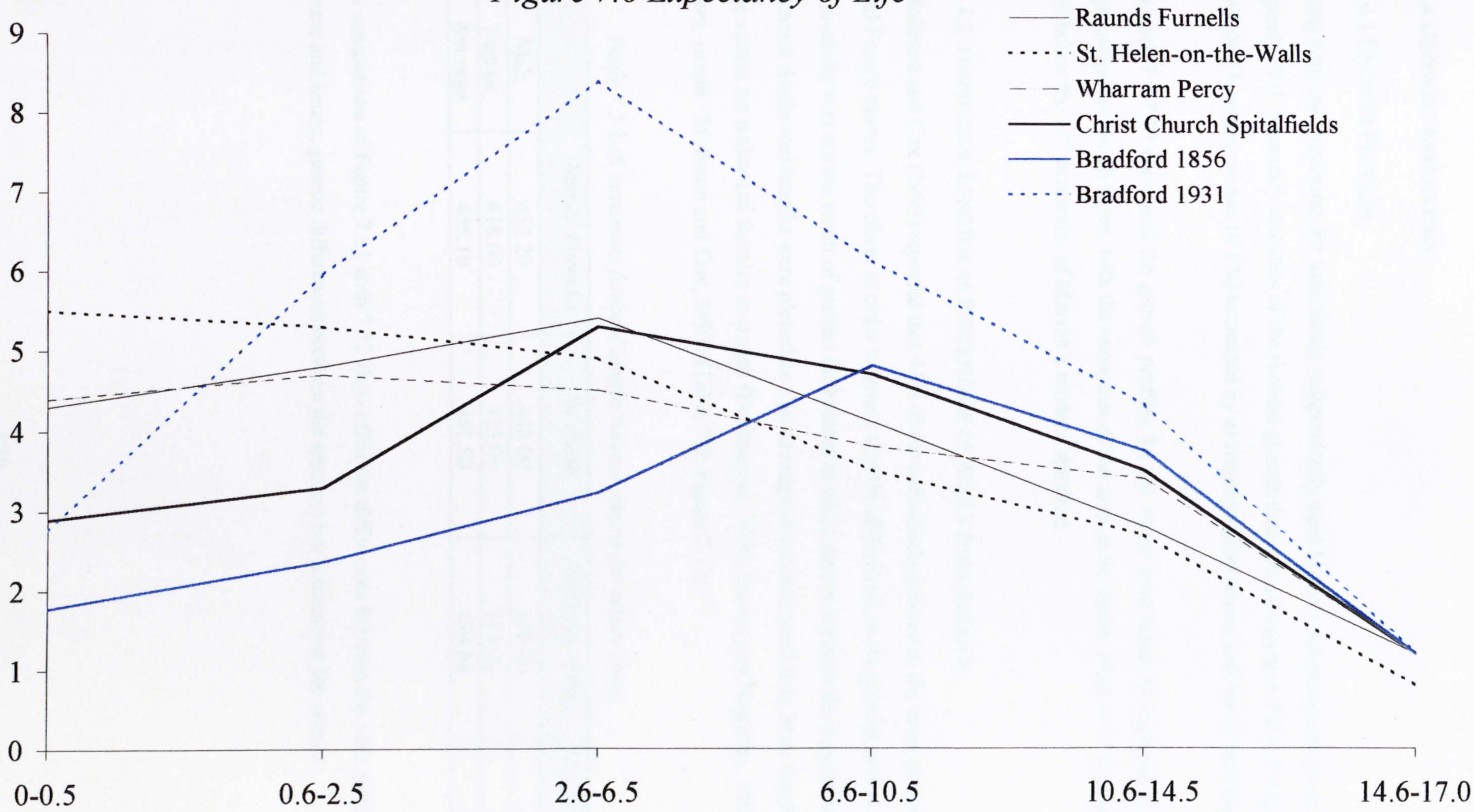


Figure 7.6 Expectancy of Life



7.4 GROWTH ESTIMATION

7.4.1 GROWTH PROFILES

Long bone measurements for individuals independently aged by their dentitions are given in Appendix VII. Summary statistics of the skeletal growth data are presented in Table 7.4 (p131) and include 25 measurements (8.1%) estimated by averaging values above and below the missing data.

Figures 7.7-7.11 compared the growth profiles for the major long bones of each site in one-year age categories. In all cases, with the exception of the ulna in St. Helen (Figure 7.9), these profiles fell below the 50th percentile of Maresh's modern standard.

7.4.2 DIAPHYSEAL LENGTHS AS PERCENTAGE OF ADULT BONE LENGTH

Molleson and Cox (1993) reported that 44% of the individuals interred in the crypt at Spitalfields had French names. Therefore, in order to insure that the differences in the growth curves of the non-adults was not the result of genetic differences in adult stature between the populations, the femoral diaphyseal lengths were plotted as a percentage of attained adult long bone length, with the values for males and females averaged (Boddington, 1996; Dawes and Magilton, 1980; Mays *pers. comm.*; Molleson and Cox, 1993) (Table 7.5; Figure 7.12).

Table 7.5 Left maximum femoral length measurements for adults (mm)

	Raunds Furnells	*St. Helen*	*Wharram Percy*	*Spitalfields*
Male	452.20	448.00	448.00	451.00
Female	418.00	415.00	413.00	417.10
Average	**435.10**	**431.50**	**430.50**	**434.10**

A comparison of Figure 7.11 with 7.12 shows that the differences between the sites remained the same and hence, genetic differences between the sites are not influencing the results.

Table 7.4

The range of long bone lengths (mm) for each age category in each site was constructed using a non-parametric t-distribution of Student's t test to obtain a 95% confidence interval. In some cases, there was only a single measurement for a specific bone in the age category or the confidence interval was too large to allow an age range to be constructed.
s.d. = standard deviation.

Humerus

	Raunds Furnells					St Helen-on-the-Walls					Christ Church Spitalfields					Wharram Percy				
				95% confidence					95% confidence					95% confidence					95% confidence	
	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range
0-0 5	4	83 10	13 87	22 05	(61 05-105 15)	2	80 60	3.11	28 20	(52 40-108 80)	16	76 20	9 41	5 00	(71 19-81 20)	17	80 90	6 80	3 49	(77 4-84 4)
0 6-2.5	14	124 61	16 54	9 55	(115 06-134 16)	11	118 30	14 73	9 92	(108 38-128 22)	41	102 10	13.30	4 20	(97 89-106.30)	21	112 00	15 80	7 33	(104 6-119 3)
2 6-6 5	14	155.64	20 69	11 94	(143 70-167.58)	23	162 30	17.30	7 47	(154 80-169 70)	20	145 80	16.00	7.48	(138 31-153 28)	45	162 00	20 00	6 01	(155.9-168)
6 6-10 5	10	199 10	26 51	18 94	(180 16-218 04)	15	203 00	23 65	13 07	(189.93-216 07)	10	201 80	2 00	17 15	(184 64-218 90)	15	211 00	17.40	9 54	(201 5-220 5)
10 6-14 5	7	246 29	22 01	20 43	(225 90-266 72)	7	243 86	14 96	13 89	(230 00-257.70)	3	235 67	24 01	65.91	(169.75-301.58)	5	237.50	20 70	26 16	(211 3-263.65)
14 5-17.0	4	252 00	13 37	21 24	(230.76-273 24)	0	-	-	-	-	0	-	-	-	-	1	247 00	0 00	-	-

Radius

	Raunds Furnells					St Helen-on-the-Walls					Christ Church Spitalfields					Wharram Percy				
				95% confidence					95% confidence					95% confidence					95% confidence	
	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range
0-0 5	3	64.00	8 41	20 89	(43.10-84 89)	1	64 70	-	-	-	19	59 18	6.04	2 89	(56 28-62 07)	7	62 80	2.50	2 35	(60 4-65 15)
0 6-2 5	13	88 54	10 31	3 24	(82 29-94 78)	4	88 43	10 31	16.37	(72 02-104.70)	34	77.15	10 69	3.73	(73 41-80 80)	13	81 90	10 70	6 47	(75 43-88.37)
2 6-6.5	10	113 80	15 17	10 85	(102 95-124 65)	13	122 19	11 65	7 05	(115 12-129 24)	19	106 26	11.75	5 67	(100 59-111 90)	34	131.90	14 50	5.10	(126 8-137)
6 6-10.5	8	144 88	18 75	15 62	(129 26-160 50)	12	150 67	18 41	11.70	(138 96-162 37)	10	146 10	19.27	13 76	(132 33-159 86)	10	150 90	13 10	9 24	(141 6-160 14)
10 6-14 5	5	173.80	11.30	14 01	(159 79-187 81)	5	185 20	19 36	24 13	(161 07-209 30)	2	172 00	14 14	127.35	*(44 70-299 35)*	4	181 30	10 60	16 85	(164 4-198 15)
14 5-17.0	2	180 50	7.78	70.16	(110.34-250 66)	0	-	-	-	-	1	184 00	-	-	-	3	193.70	12.70	31 86	(161 8-225 5)

Ulna

	Raunds Furnells					St Helen-on-the-Walls					Christ Church Spitalfields					Wharram Percy				
				95% confidence					95% confidence					95% confidence					95% confidence	
	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range
0-0.5	3	72.20	8 41	28 72	(43 47-100 92)	1	73 40	-	-	-	17	66 06	5 66	2 90	(56 28-68 96)	6	71.70	3 50	3 75	(67 9-75.4)
0 6-2 5	10	99 81	9 89	7.07	(92 70-106 88)	5	95.66	10 58	13 18	(82 48-108 84)	29	82.59	9 50	3 61	(78 98-86.20)	11	92.40	8 80	5 93	(86 4-98 3)
2.6-6.5	12	125.56	15 56	9.88	(115.68-135.44)	15	134 25	13 60	7.51	(126 74-141.76)	18	115 39	13 28	13 45	(101 93-128 84)	33	131.90	14 40	5 16	(126 7-137)
6 6-10.5	9	163 33	21 31	16 40	(146 93-179 73)	14	167 50	16 31	9 42	(158 08-176 92)	10	160 00	19.20	13 71	(146 28-173.71)	10	166 90	13.40	8 36	(158 5-175.2)
10 6-14.5	4	196 50	11.50	18 28	(178 21-214 78)	1	232 00	-	-	-	3	189 33	9 87	24 51	(164 82-213 84)	4	198 30	6 50	10 33	(187 9-208 6)
14.5-17 0	3	208 67	9 45	23.48	(185 19-232.15)	0	-	-	-	-	1	196 00	-	-	-	2	220 50	30.40	275 80	-

Femur

	Raunds Furnells					St Helen-on-the-Walls					Christ Church Spitalfields					Wharram Percy				
				95% confidence					95% confidence					95% confidence					95% confidence	
	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range
0-0.5	2	89 95	0.07	0.13	(89 82-90 08)	1	99.30	-	-	-	17	91.44	12 35	6.33	(85.10-97 77)	9	98 80	12 50	9 63	(89 16-108 4)
0 6-2 5	14	150 59	25.58	14.77	(135 82-165 36)	10	155 84	17.59	15 48	(140.30-171 32)	36	130 25	19.26	6.55	(123 70-136.80)	20	139 00	23 90	11 09	(127.9-150)
2 6-6.5	14	206 88	29 17	16 85	(190 03-223-73)	19	218 37	33.22	15 98	(202 38-237.3)	19	195 26	22.70	10 96	(184 29-206.22)	31	220 10	29 10	10 66	(209 4-230 8)
6 6-10 5	10	271.80	38 32	27.41	(244 39-299 21)	15	285 07	24 49	13 54	(271 53-298 61)	8	281.75	39 89	34 00	(247.70-315.75)	12	293 80	24 10	15 31	(278 4-309.1)
10 6-14.5	6	341 33	20 26	21 33	(320 00-362 66)	7	354 29	38 41	35 62	(318 66-389 91)	5	327.40	35.15	43 81	(283.58-371.21)	6	343 60	13 60	14 55	(329-358 1)
14 5-17.0	3	347.33	18 15	45 11	(302 22-392 44)	0	-	-	-	-	1	373 00	-	-	-	3	378 00	25 20	62 80	(315 2-440 8)

Tibia

	Raunds Furnells					St Helen-on-the-Walls					Christ Church Spitalfields					Wharram Percy				
				95% confidence					95% confidence					95% confidence					95% confidence	
	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range	N	Mean	s.d.	interval	range
0-0.5	0	-	-	-	-	2	84 90	1.77	15 89	(69 01-100.79)	22	79.02	11 37	5 03	(73 99-84 05)	7	80 00	5 40	4 99	(75-84.9)
0 6-2.5	11	123 07	14 38	9 65	(113 40-132 72)	7	119 42	15 00	13 32	(106 10-132 74)	36	107 94	16 26	5.53	(102 41-113 47)	19	15 10	15 20	7 33	(107.7-122 4)
2.6-6 5	13	165 85	24 54	14 86	(150 99-180.71)	14	170 81	25 07	14 47	(156 30-185 30)	17	156.71	20 54	10 56	(146 15-167 27)	28	171 60	22 70	8 79	(162 8-180 4)
6 6-10 5	6	222.33	37 68	39 68	(182 64-262 01)	14	227 50	21.32	12 31	(215 19-239 81)	10	231.90	32 86	23 48	(208 42-255.38)	11	233 80	23 70	15 92	(217 9-249 7)
10 6-14 5	5	270 60	15 88	18 88	(251 72-289.48)	6	286 00	33.79	35.59	(250.40-321 59)	5	288 60	48 13	59 71	(228 88-348.31)	6	269.20	13 70	14 42	(254 8-283 6)
14 5-17 0	2	299 00	36 77	331 45	-	0	-	-	-	-	1	300 00	-	-	-	3	289 00	16 50	41 02	(247 9-330)

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Figure 7.7 Humerus

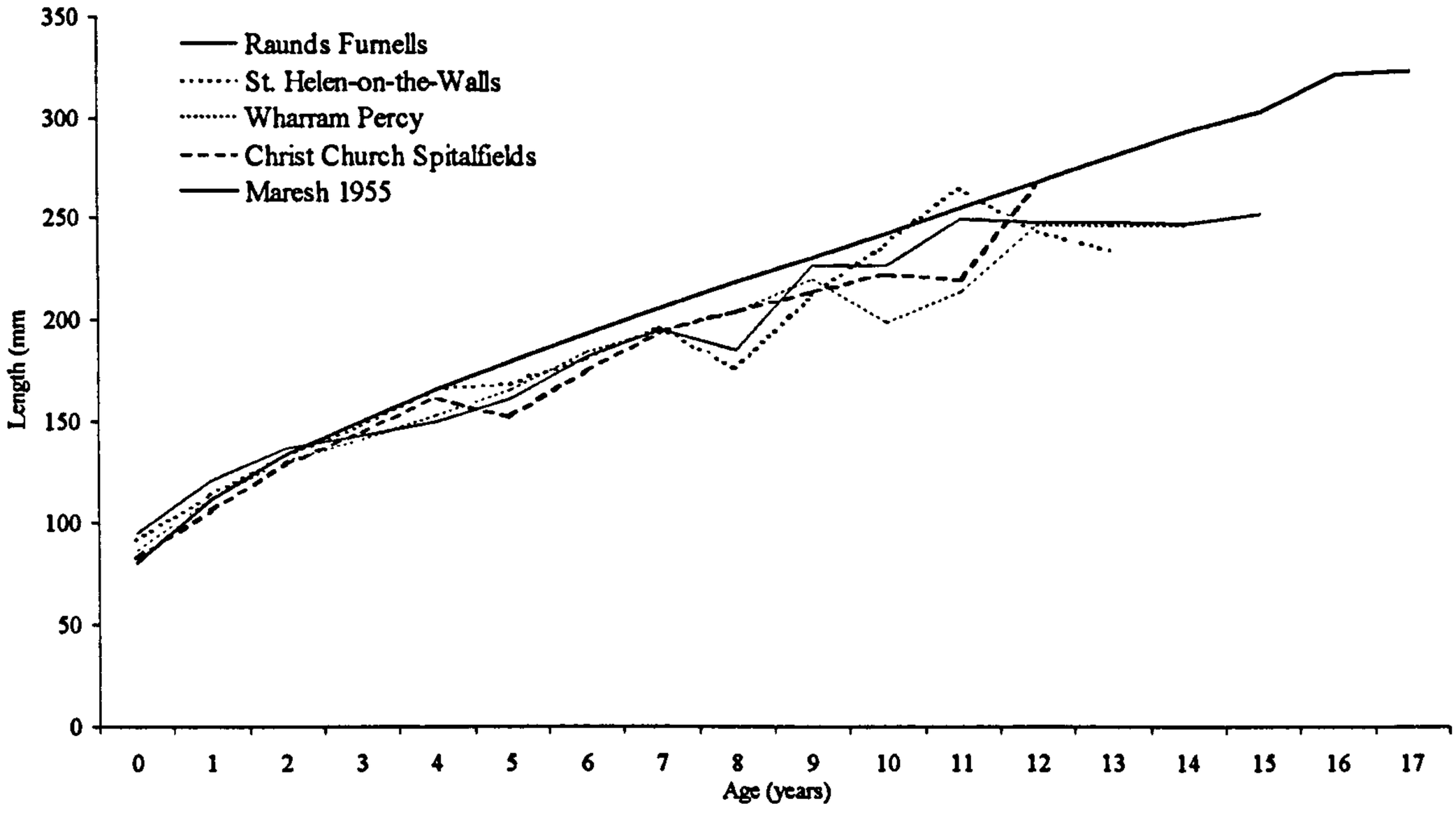


Figure 7.8 Radius

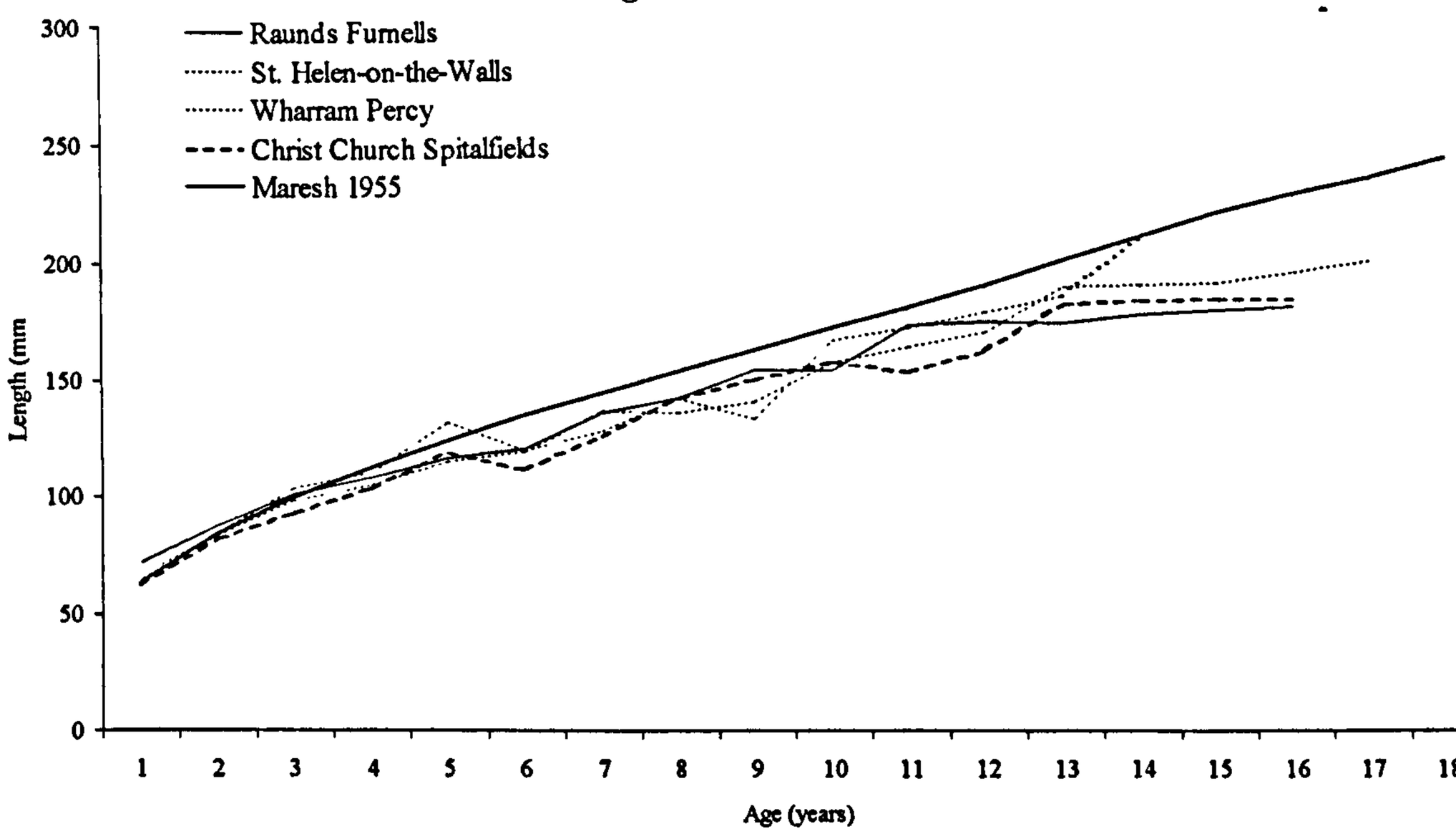


Figure 7.9 Ulna

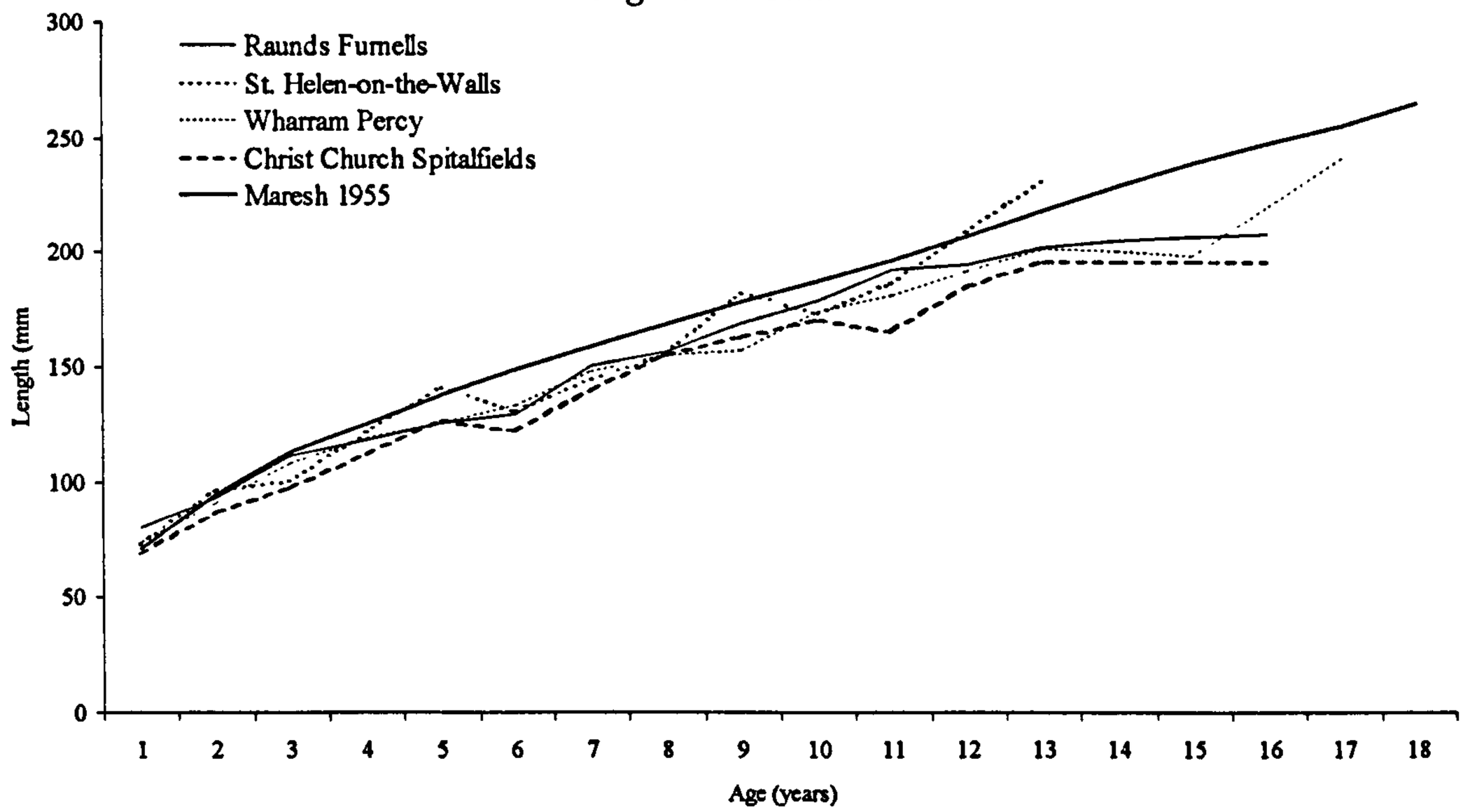


Figure 7.10 Tibia

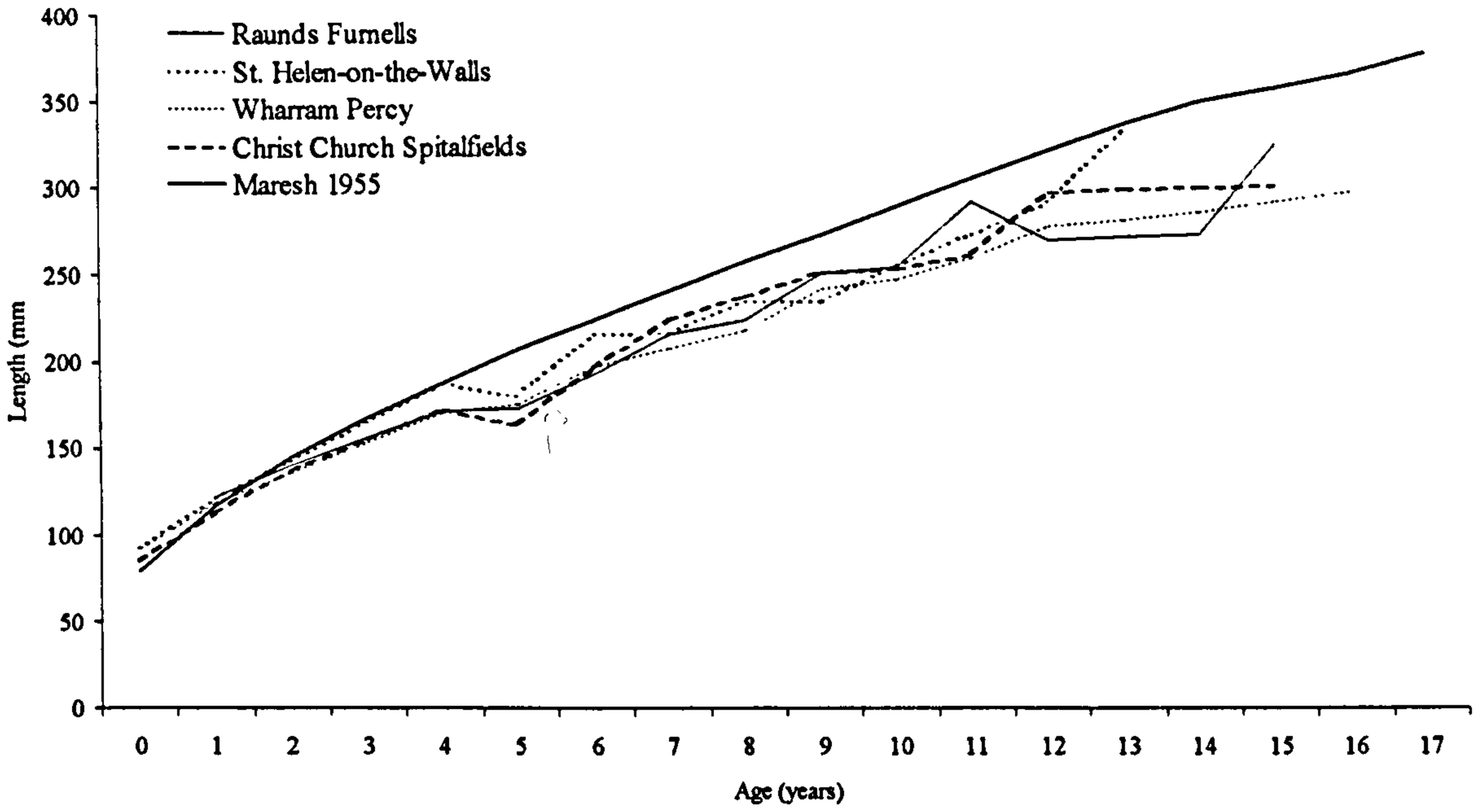


Figure 7.11 Femur

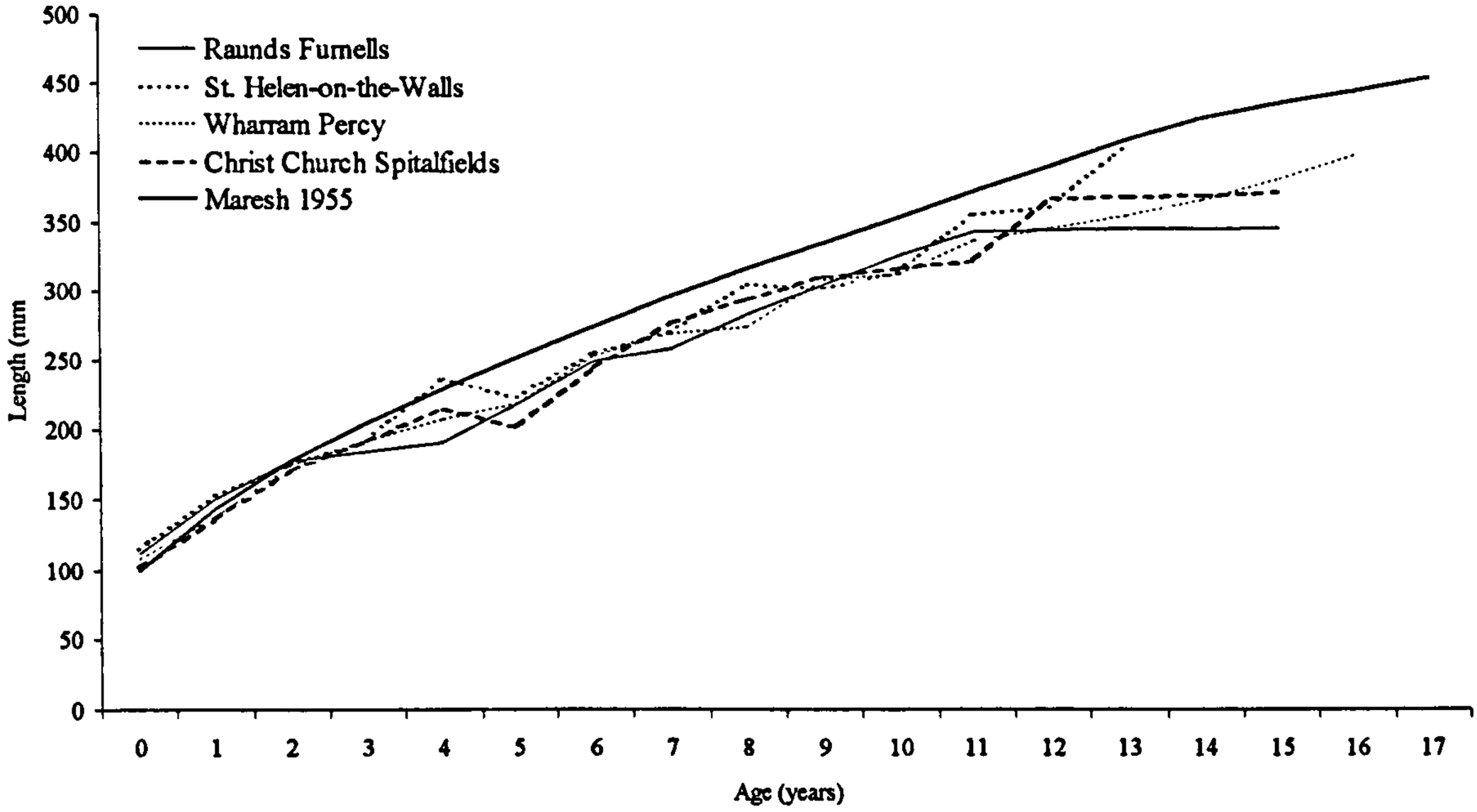
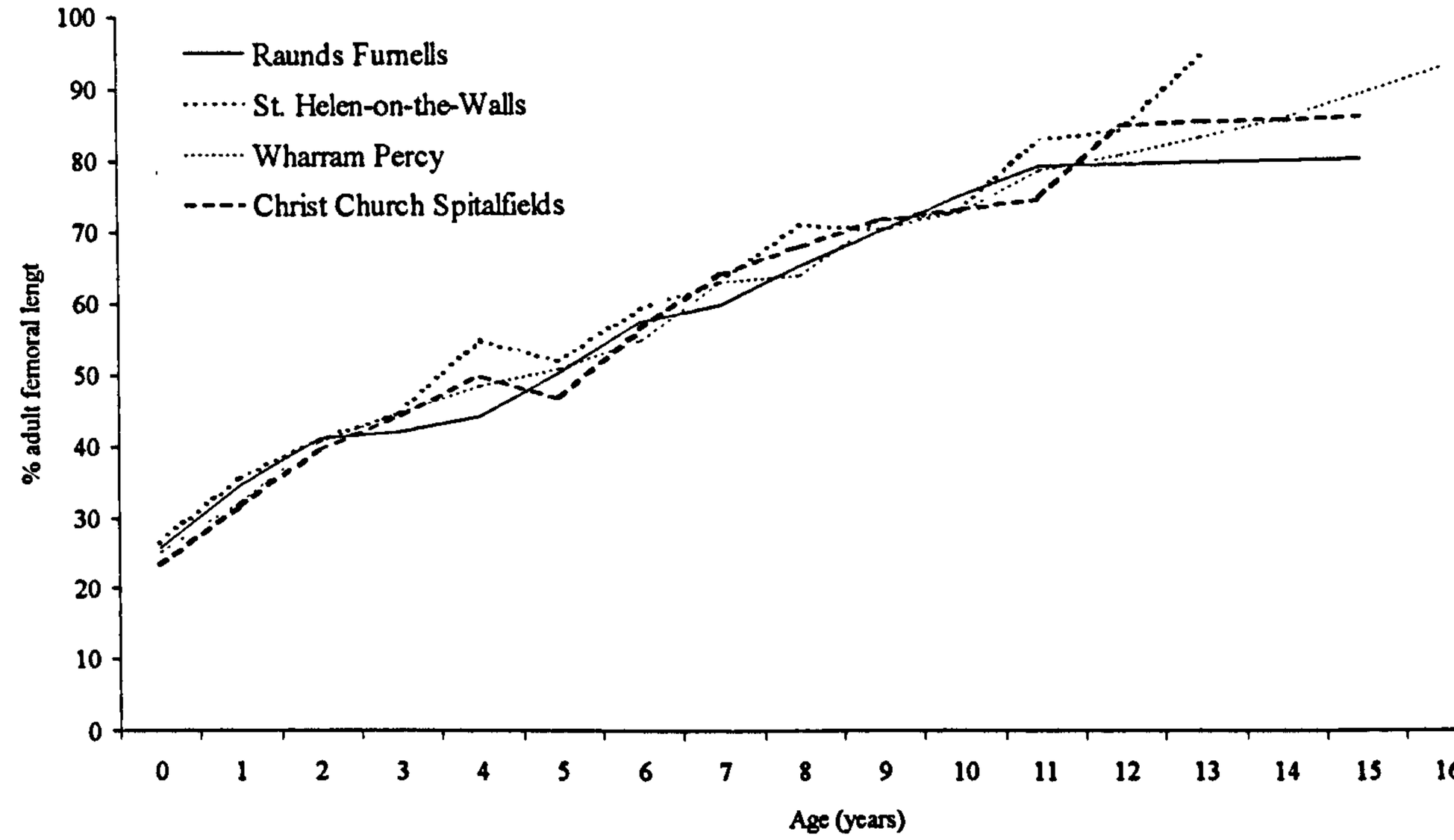


Figure 7.12 %Attainment of Mean Adult Bone Length: Femur



7.4.3 Comparison of Skeletal Growth Profiles

As growth is influenced by different environmental factors, it was of interest to see if any differences between early and late medieval rural sites, urban and rural later medieval sites and the pre-industrial and industrial sites existed. Femoral lengths were plotted as a percentage of adult attained height and two-tailed Kolmogorov-Smirnov tests were applied. Individual values (mean age/mean long bone length) for each age cohort were superimposed onto the graphs.

7.4.3.1 The Rural Sites

Figure 7.13 shows the growth differences between Raunds Furnells (Anglo-Saxon) and Wharram Percy (later medieval). During infancy the Anglo-Saxon children appeared to be larger than the later medieval children (by 1.2 cm) but fell below their later counterparts around two years of age (by 1.6 cm) and, again, from 12 years of age. However the values for the adolescent age groups are estimated (see Table 7.4) and the two patterns of growth were not statistically significant at the 95% confidence interval. The delay in growth between the ages of two and four years at Raunds Furnells is probably due to small sample sizes at four years (n=3) and an estimated value at three years.

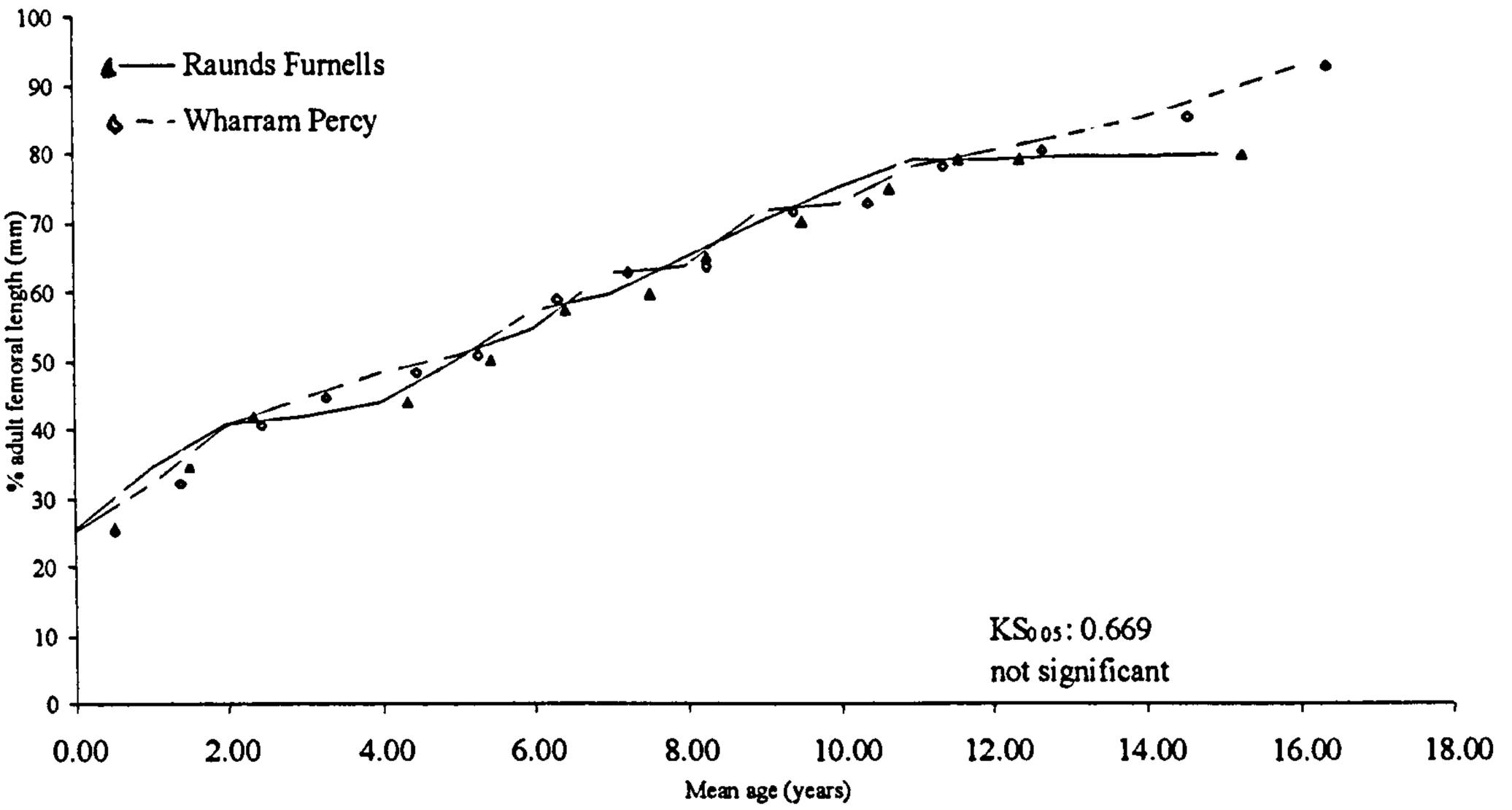


Figure 7.13 Comparison of early and later medieval sites

7.4.3.2 The Later Medieval Sites

A comparison of Wharram Percy's and St. Helen's femoral diaphyseal length profiles showed that the urban children were generally larger than their rural peers throughout the growth period. They were 2.9 cm taller at four years, and 3.1 cm taller in the eight-year age cohort (Figure 7.14). However, these differences were not significant. As with the previous graph, the two curves began to deviate at 12 years of age, but as it was not possible to sex these individuals, differences in the proportion of males and females at these sites may be influencing the curves.

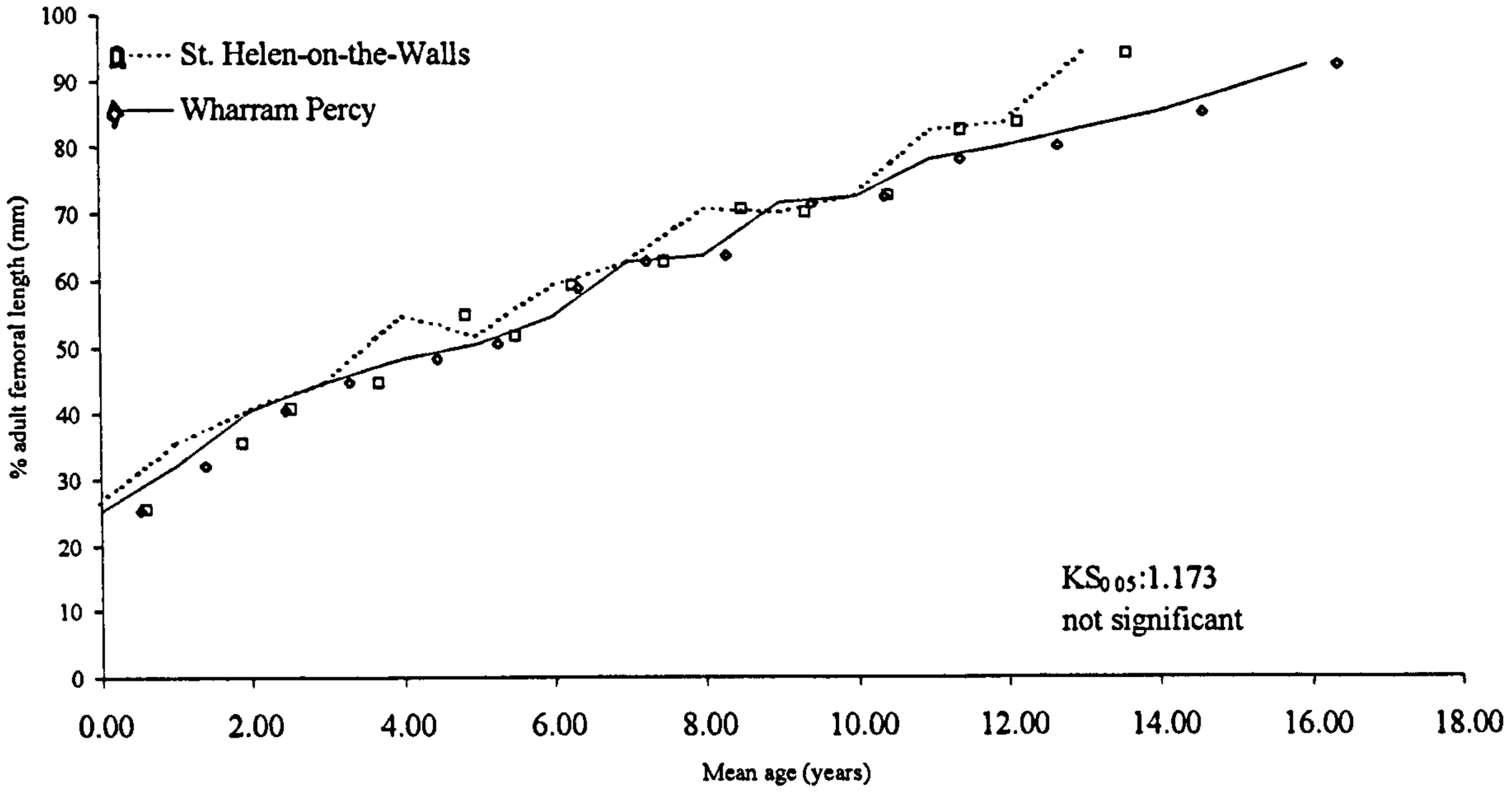


Figure 7.14 Comparison of later medieval urban and rural sites

7.4.3.3 The Pre-Industrial and Industrial Sites

When the pre-industrial and industrial sites (St. Helen and Spitalfields) were compared (Figure 7.15) the growth profile of the Spitalfields children fell significantly below that of their medieval peers. Between 0-1 year of age the Spitalfields children were an average of 1.5 cm shorter than their medieval counterparts, 2.1cm shorter between the ages of four and five years, and 3.4 cm smaller at 11 years; this difference may be result of growth spurt.

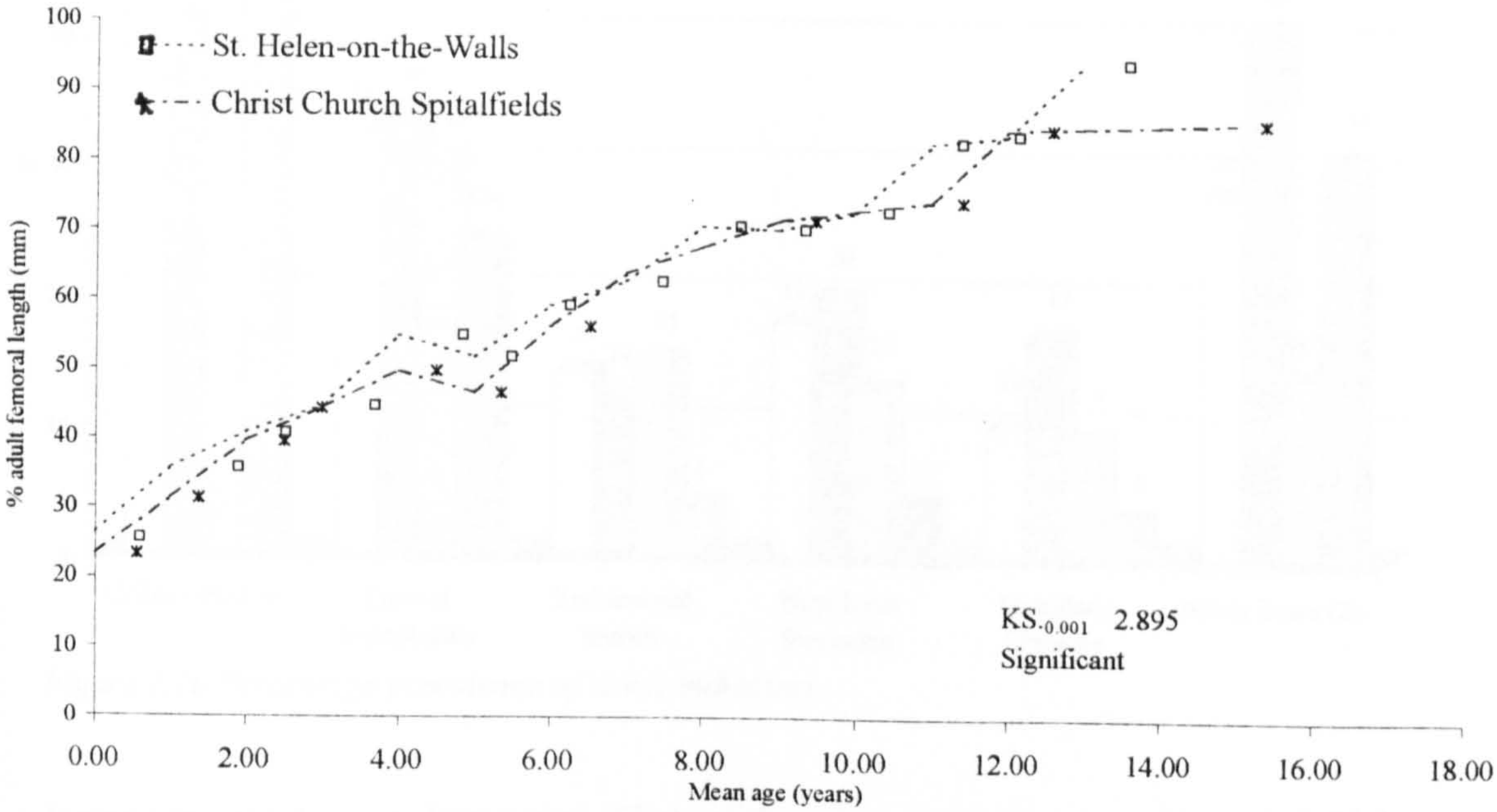


Figure 7.15 Comparison of pre-industrial and industrial sites

7.5 INDICATORS OF NON-SPECIFIC STRESS

7.5.1 PREVALENCE OF STRESS INDICATORS BETWEEN THE SITES

Detailed tables showing the number and (true) percentage of stress indicators in each sample are provided in Appendix VIII. Table 7.6 and Figure 7.16 present the summary statistics. The chi - squared tests for 2 x 2 and 2 x *c* tables, with Yate's continuity correction (Kirkwood, 1988), were carried out to test the differences in prevalence of stress indicators between the sites.

Table 7.6 True prevalence of indicators of stress in each site: individuals affected (aged only).

Stress Indicator	*Raunds Furnells*	*St. Helen*	*Wharram Percy*	*Spitalfields*
	% (*n*)	% (*n*)	% (*n*)	% (*n*)
Cribra orbitalia	55 (46)	56 (49)	56 (112)	57 (63)
Enamel hypoplasias	32 (23)	34 (31)	30 (36)	24 (22)
Endocranial lesions	14 (14)	12 (11)	15 (33)	4 (5)
New bone formation	18 (15)	20 (21)	13 (21)	4 (6)
Maxillary sinusitis	10 (2)	17 (6)	9 (8)	3 (1)
Harris lines (grade 2)	28 (16)	41 (23)	14 (19)	31 (14)

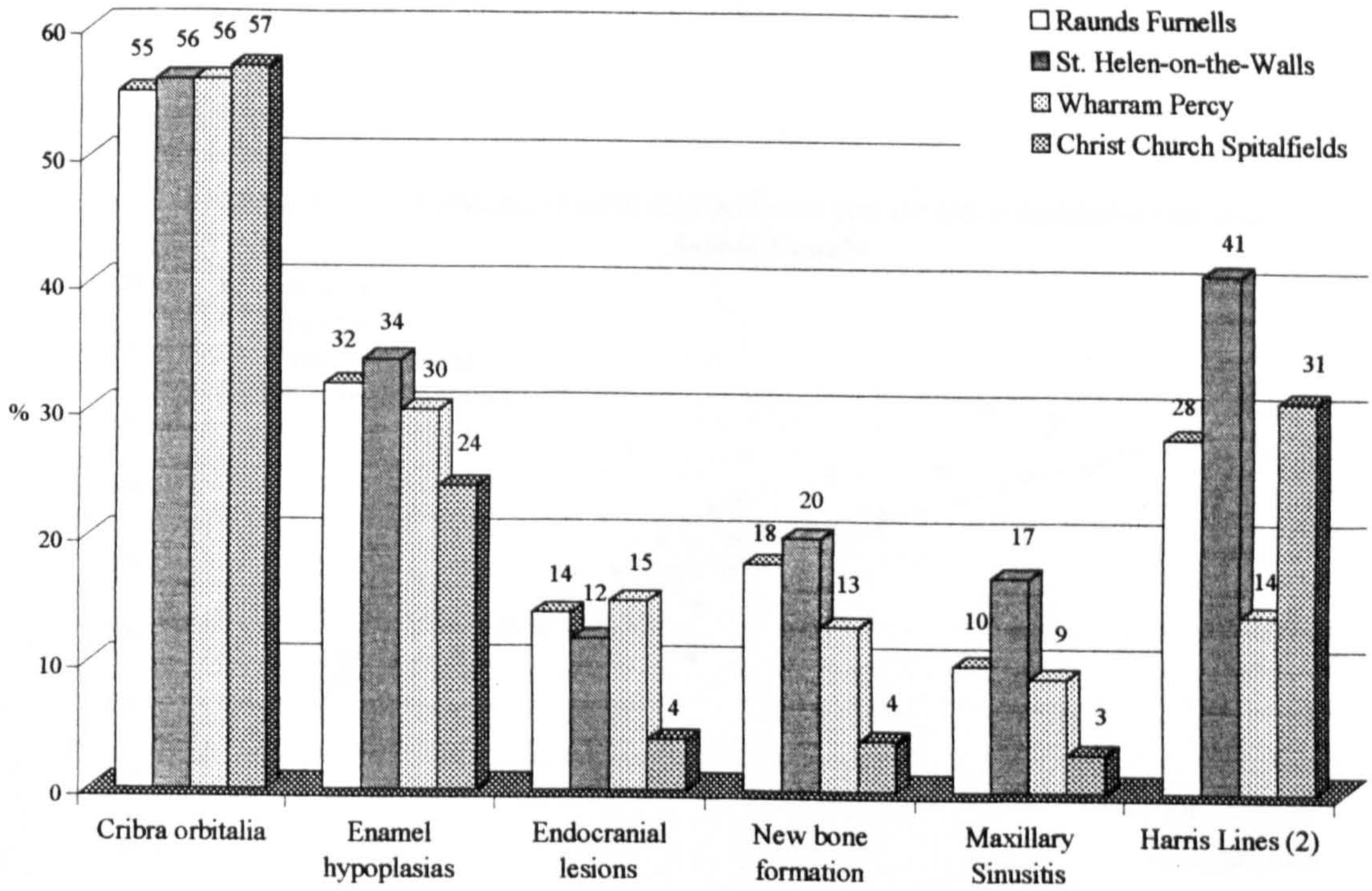


Figure 7.16 Percentage prevalence of stress indicators

It was expected that any detrimental effects of the urban environment would result in higher prevalences of stress indicators in the children from St. Helen and Spitalfields. However, chi-squared tests revealed that there were no significant differences between the sites, although the children at St. Helen had the highest prevalence of periostitis, enamel defects and sinusitis. In fact at Spitalfields, the children had significantly less periostitis than at the other sites. The prevalence of stress indicators between sites and between different age groups will be presented in more detail in the following sections.

7.5.2 The Impact of Stress on Growth

The femoral diaphyseal lengths of individuals with one or more indicators of stress were plotted against those without any skeletal evidence of stress, and linear regression lines were added (Figures 7.17-7.20). The lower limbs are regarded as the most sensitive to environmental stress as they represent the fastest growing parts of the body during childhood (Eveleth and Tanner, 1990). Therefore, femoral values were used to produce the scatter plots. The differences between the two distributions were tested using the two-tailed Kolmogorov-Smirnov statistic. Tests revealed that stress indicators did not have a significant impact on the growth of the 'stressed' individuals at any of the sites with the exception of Wharram Percy, where the 'stressed' individuals were generally taller than those who showed no sign of stress.

Figure 7.17 Individuals with and without one or more indicators of stress
Raunds Furnells

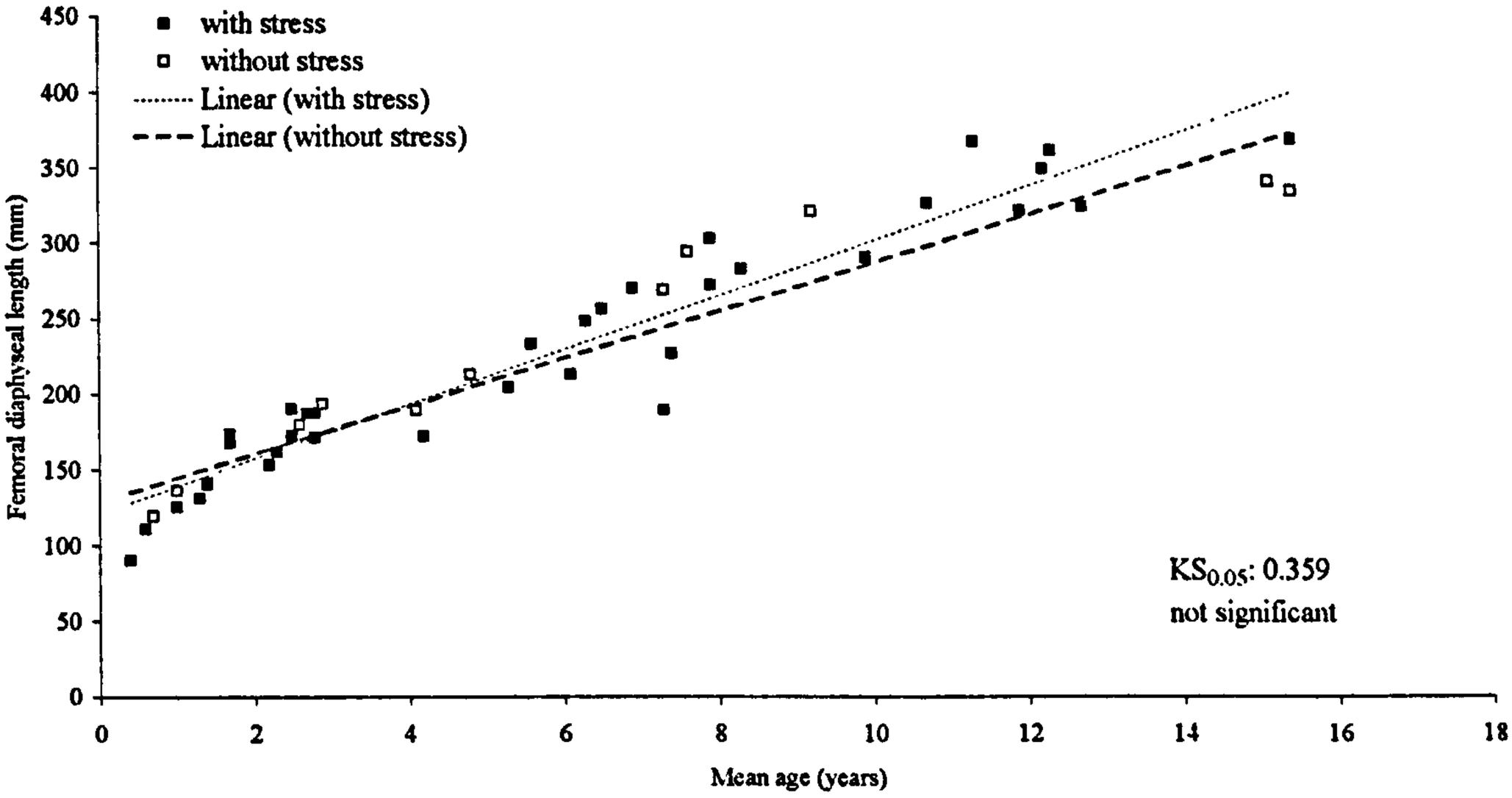


Figure 7.18 Individuals with and without one or more indicators of stress
St. Helen-on-the-Walls

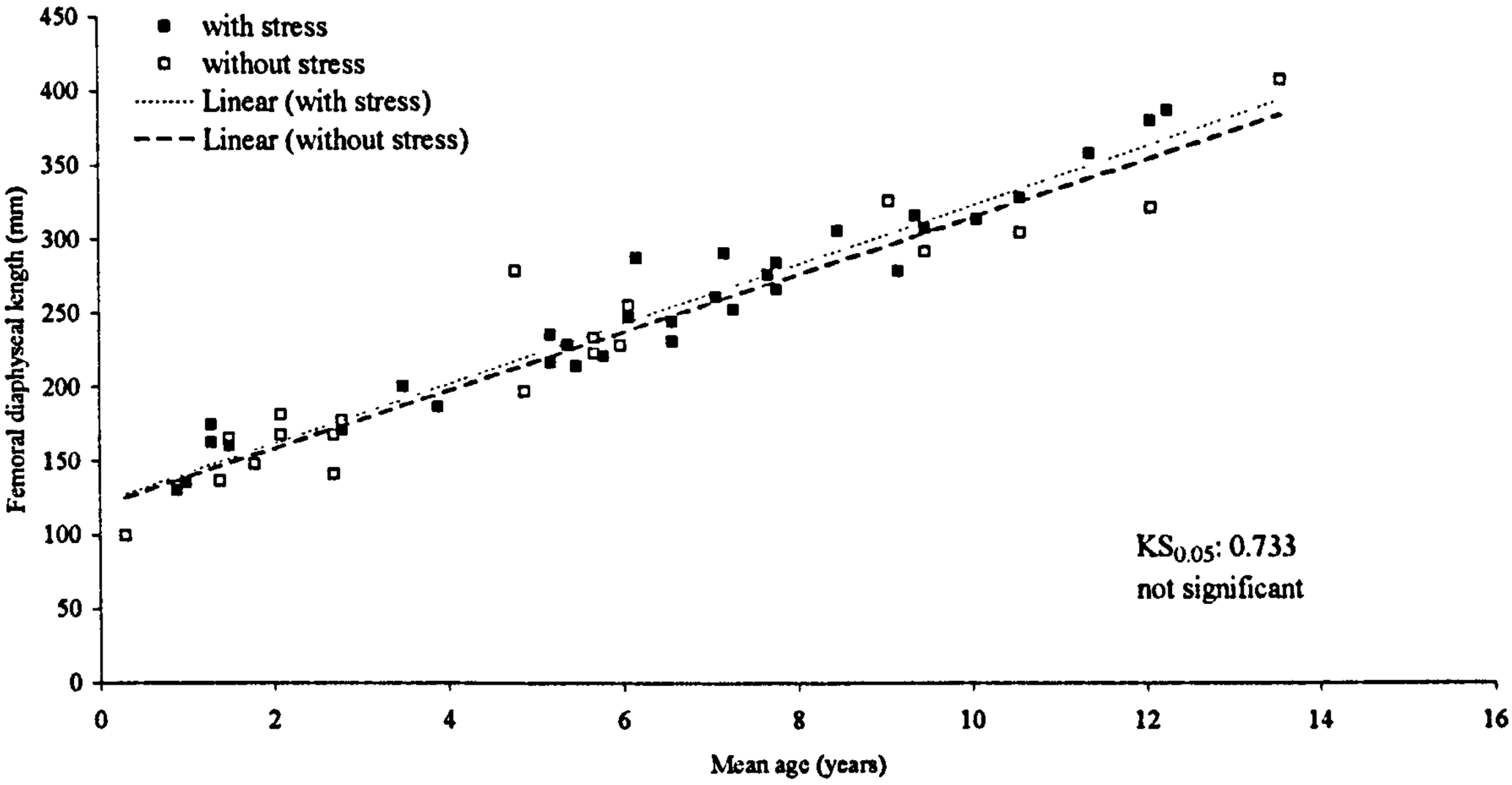


Figure 7.19 Individuals with and without one or more indicators of stress
Wharram Percy

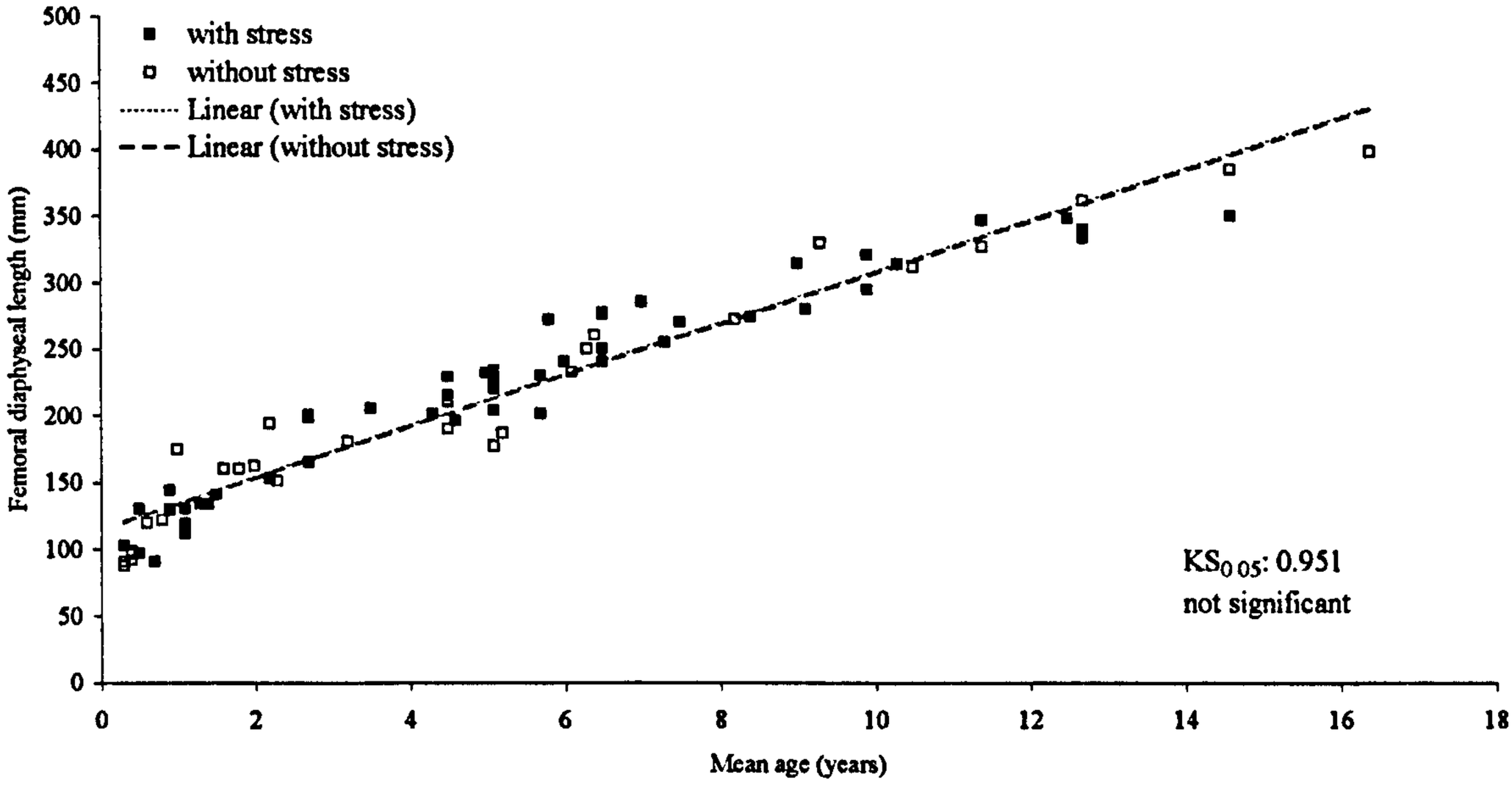


Figure 7.20 Individuals with and without one or more indicators of stress
Christ Church Spitalfields

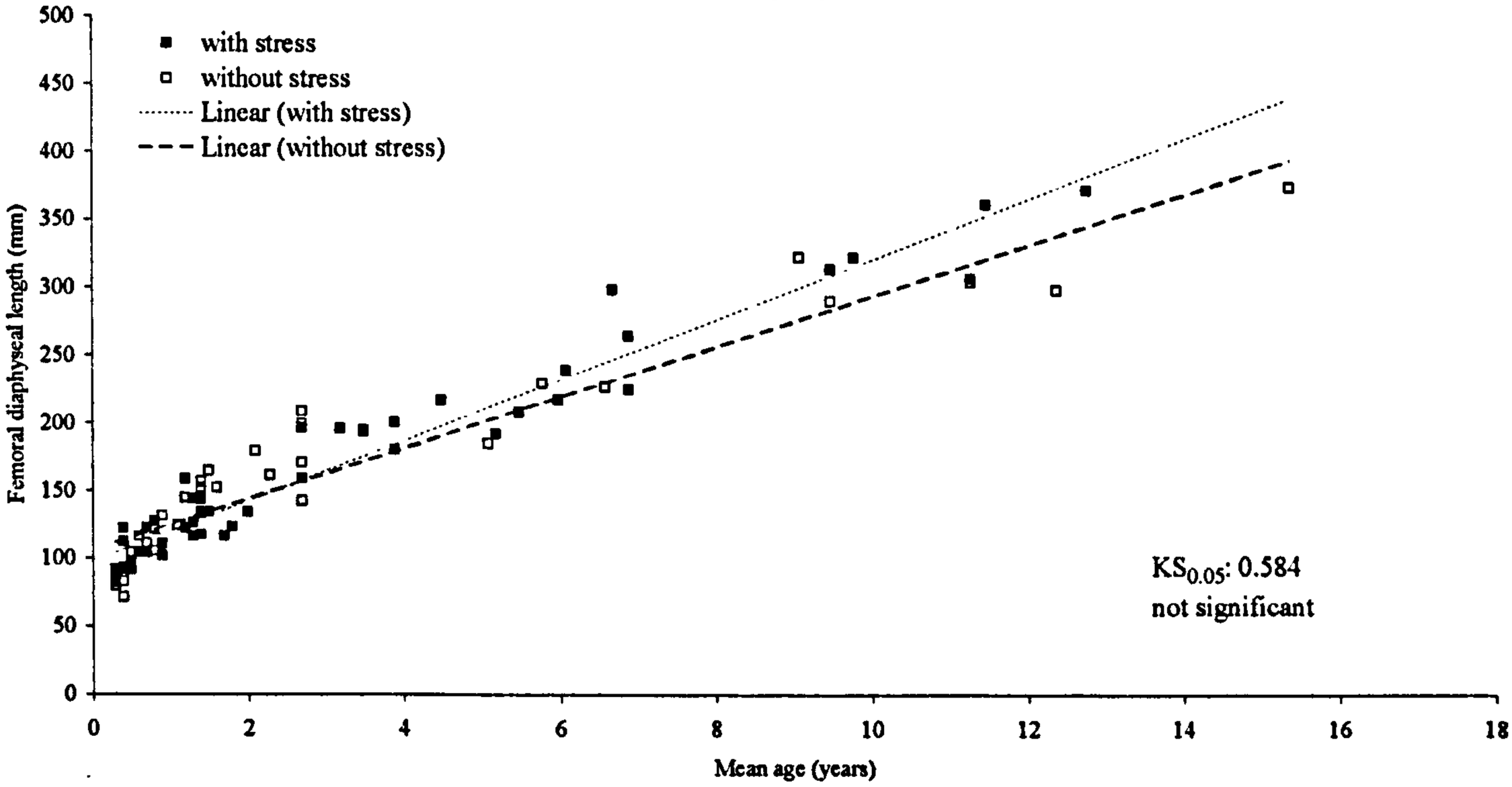


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Figure 7.21 Individuals with new bone formation: ***Raunds Furnells***

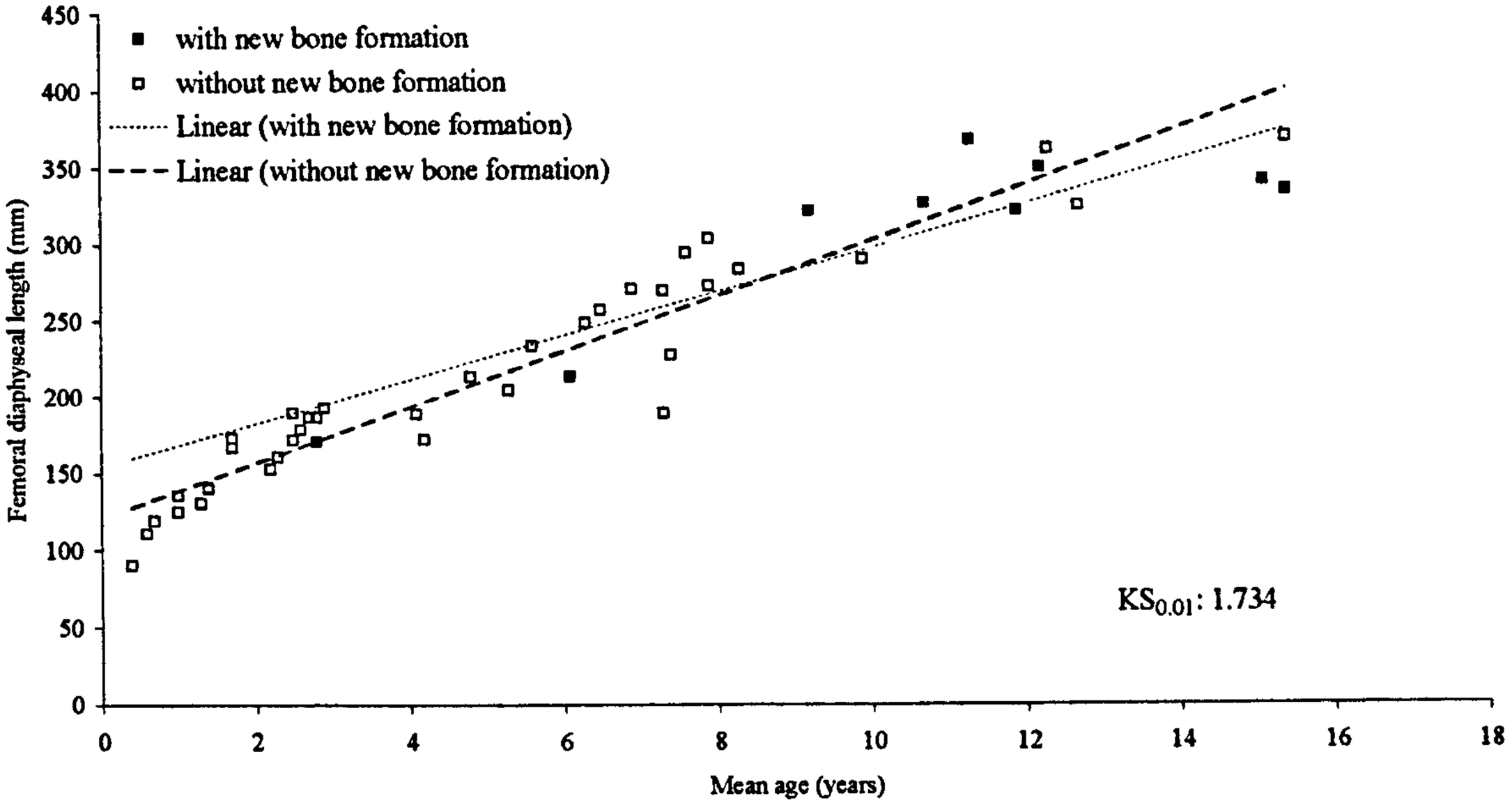


Figure 7.22 Individuals with cribra orbitalia: ***St. Helen-on-the-Walls***

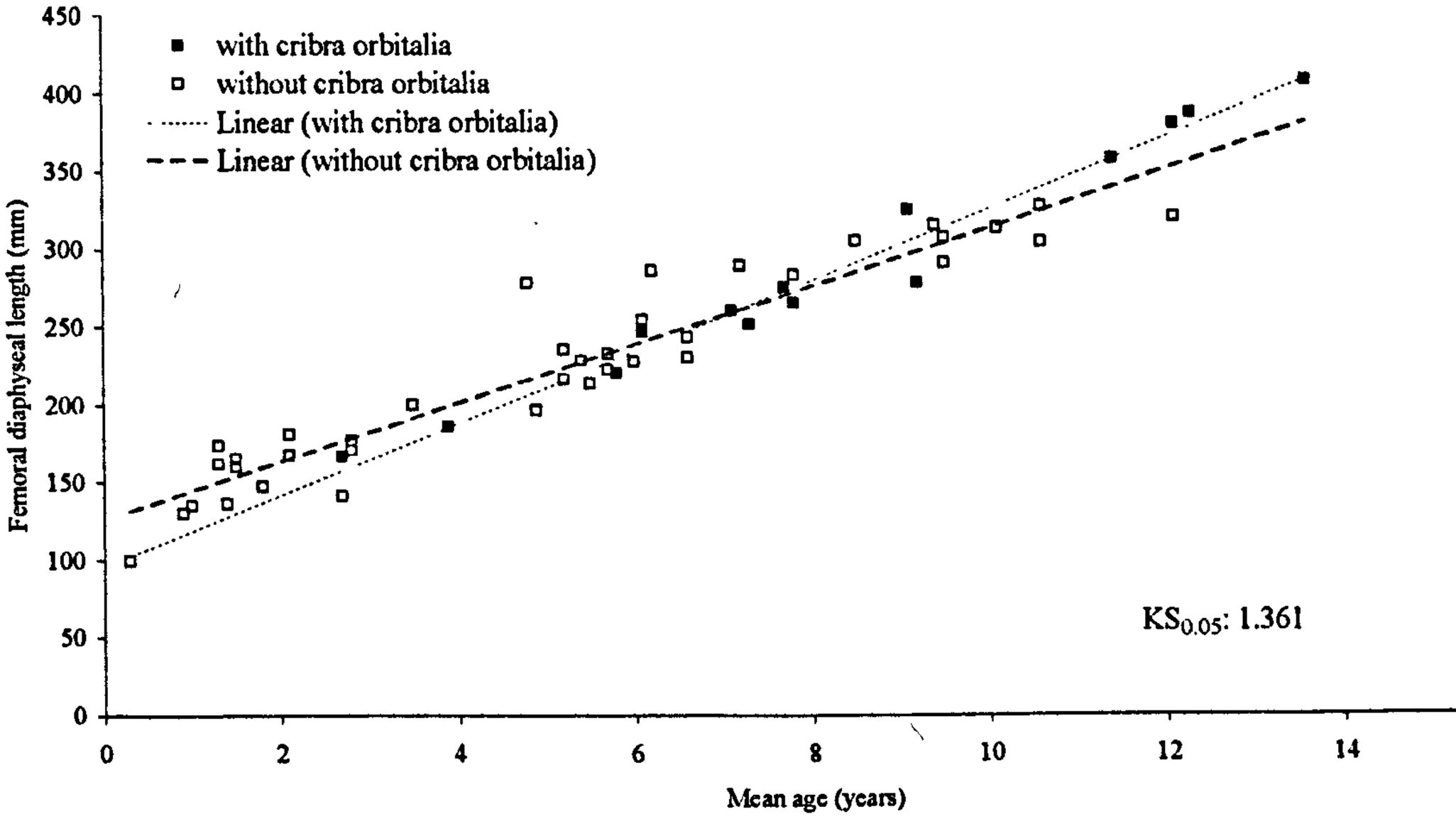


Figure 7.23 Individuals with cribra orbitalia: ***Christ Church Spitalfields***

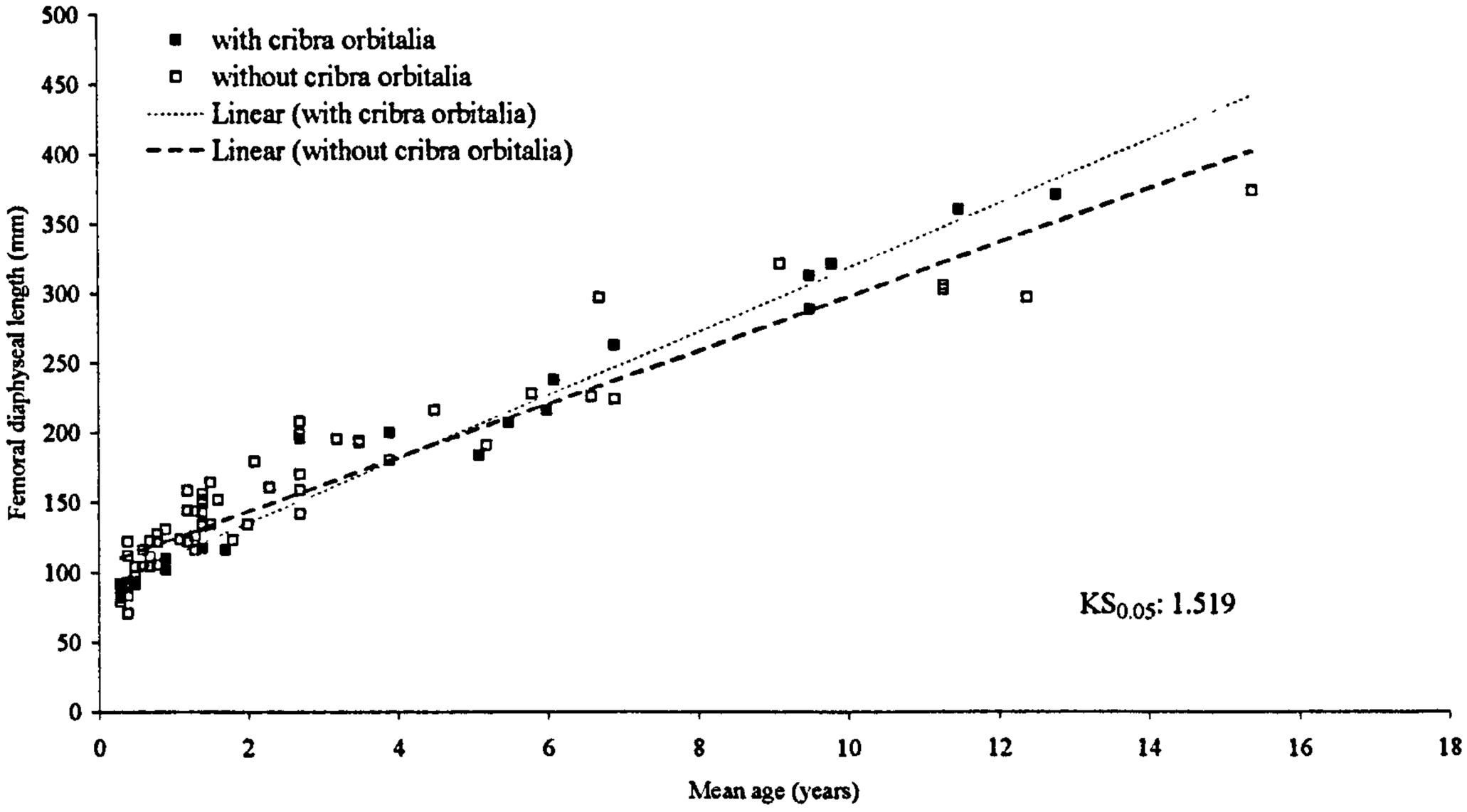
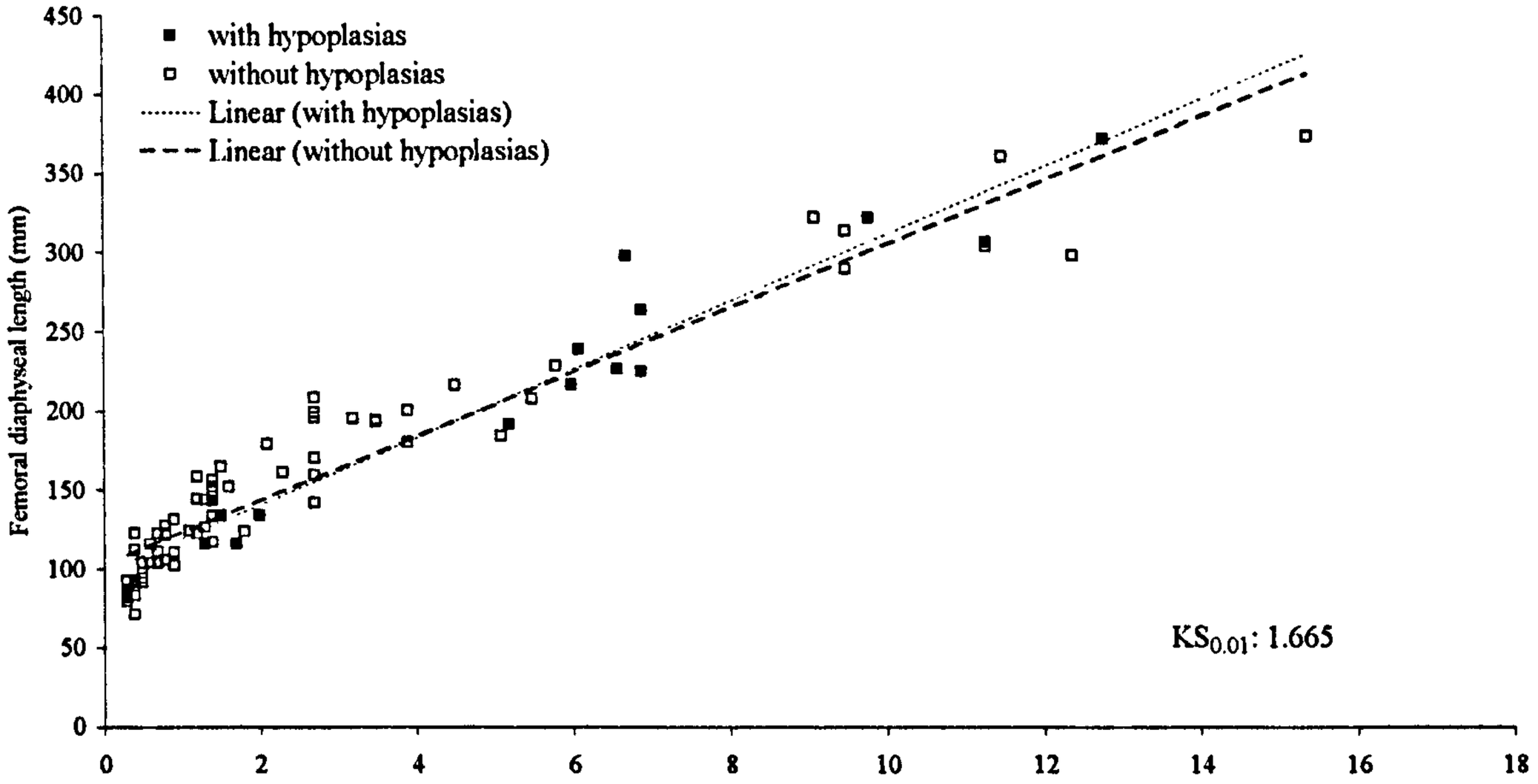


Figure 7.24 Individuals with enamel hypoplasias: ***Christ Church Spitalfields***



At both St. Helen (Figure 7.22) and Spitalfields (Figure 7.23), cribra orbitalia had an effect on the growth profiles of the non-adults. At St. Helen, the children in the five-year age category with cribra orbitalia were 0.4 cm smaller than their non-affected peers but were taller at seven years by 2.4 cm and by 6 cm at 12 years. At Spitalfields, children with orbital lesions were generally smaller than the children without lesions until the later age categories, where numbers become small and comparisons more difficult. However, the growth profiles follow a similar pattern to that at St. Helen with the adolescent children with cribra orbitalia becoming taller than those without orbital lesions. The differences in growth profiles of the 'stressed' and 'non-stressed' at Wharram Percy, evident in the initial analysis do not appear to be the result of any one particular indicator.

Despite being associated with a cessation of growth following infection or malnutrition, Harris lines did not have an impact on the growth of the non-adults, and enamel hypoplasias only affected the growth of the non-adults at Spitalfields (Figure 7.24). In the one-year age cohort, the children with hypoplasias were 1 cm smaller then those with no defects, but by 18 years showed a trend to be taller than their unaffected peers.

7.5.3 Mean Age at Death With and Without Stress Indicators

Numerous studies of skeletal and living adult populations have shown that lesions such as cribra orbitalia and, in particular, enamel hypoplasias are associated with increased mortality and a shorter life span. Therefore, the mean ages at death of non-adults with and without stress indicators were calculated (Table 7.8, Figures 7.25-7.28) and the cumulative age distributions tested using the Kolmogorov-Smirnov statistic.

None of the results showed that enamel hypoplasias have had a detrimental effect on the life expectancy of the children. In fact, in all cases, children with defects lived slightly longer than those without them, again, reflecting the need for the child to survive a stress episode for the defects to become visible.

Table 7.8 Mean age at death of individuals with and without indicators of stress

	Raunds Furnells					St. Helen-on-the-Walls				
	with		*without*		*difference (yrs)*	*with*		*without*		*difference (yrs)*
Cribra orbitalia	5.47	± *3.80*	4.78	± *4.95*	**0.96***	5.74	± *3.84*	5.26	± *3.33*	**0.48**
Enamel hypoplasias	9.75	± *3.49*	7.68	± *2.60*	**2.07**	8.56	± *2.48*	8.02	± *2.53*	**0.54**
Endocranial lesions	2.60	± *3.19*	5.81	± *4.26*	**2.58***	2.70	± *3.10*	4.46	± *4.27*	**1.76**
Non-specific infection	9.15	± *4.00*	5.21	± *4.10*	**3.90**	5.24	± *3.58*	5.96	± *3.93*	**0.72**
Sinusitis	10.35	± *6.72*	7.18	± *4.88*	**3.17**	8.12	± *3.60*	6.93	± *3.82*	**1.19**
Harris lines	6.34	± *4.25*	6.11	± *5.20*	**0.20**	6.02	± *3.99*	7.20	± *3.81*	**1.18**

	Wharram Percy					Christ Church Spitalfields				
	with		*without*		*difference (yrs)*	*with*		*without*		*difference (yrs)*
Cribra orbitalia	4.75	± *3.63*	3.46	± *3.74*	**1.3***	3.50	± *3.43*	2.63	± *3.54*	**0.87**
Enamel hypoplasias	7.83	± *2.93*	7.33	± *3.43*	**0.50**	8.52	± *3.32*	7.96	± *3.50*	**0.56**
Endocranial lesions	2.9	± *2.69*	4.55	± *3.91*	**1.75**	1.74	± *1.22*	2.92	± *3.42*	**1.18**
Non-specific infection	4.32	± *3.73*	4.68	± *4.10*	**0.36**	7.48	± *4.76*	2.48	± *3.04*	**5.00****
Sinusitis	5.60	± *2.10*	7.35	± *3.58*	**1.75**	6.95	± *8.27*	2.95	± *3.43*	**4.00**
Harris lines	4.82	± *2.31*	7.49	± *9.11*	**2.67**	1.15	± *1.11*	0.64	± *0.47*	**0.51**

*P = 0.5

**P = 0.01

Cribra orbitalia is associated with an increased life expectancy with the individuals displaying the lesions living up to one year longer than those without lesions. The mortality curves show that at Raunds Furnells (Figure 7.25) individuals without lesions have an earlier peak in mortality in the 0.6-2.5 year age category. At Wharram Percy (Figure 7.26) there is a much flatter mortality distribution for the individuals without cribra orbitalia, and those with the lesions have a peak in mortality in the 2.6-6.5 year age category. That endocranial lesions were consistently found to be associated with a lower mean age at death is not surprising if these lesions are reflecting early childhood infections, such as meningitis, but they may also be indicative of rapid growth in the cranial vault causing misdiagnosis of these lesions in the very young (see section 7.6.3). At Raunds Furnells, those with endocranial lesions had a peak mortality rate in the 0.6-2.5 years age category (Figure 7.27), but one older individual in the 10.6-14.5 years age category displayed lesions. At Spitalfields, individuals with new bone formation lived 5.00 years longer than those without evidence of infection (Figure 7.28).

Figure 7.25 Mortality curves of individuals with and without cribra orbitalia

Raunds Furnells

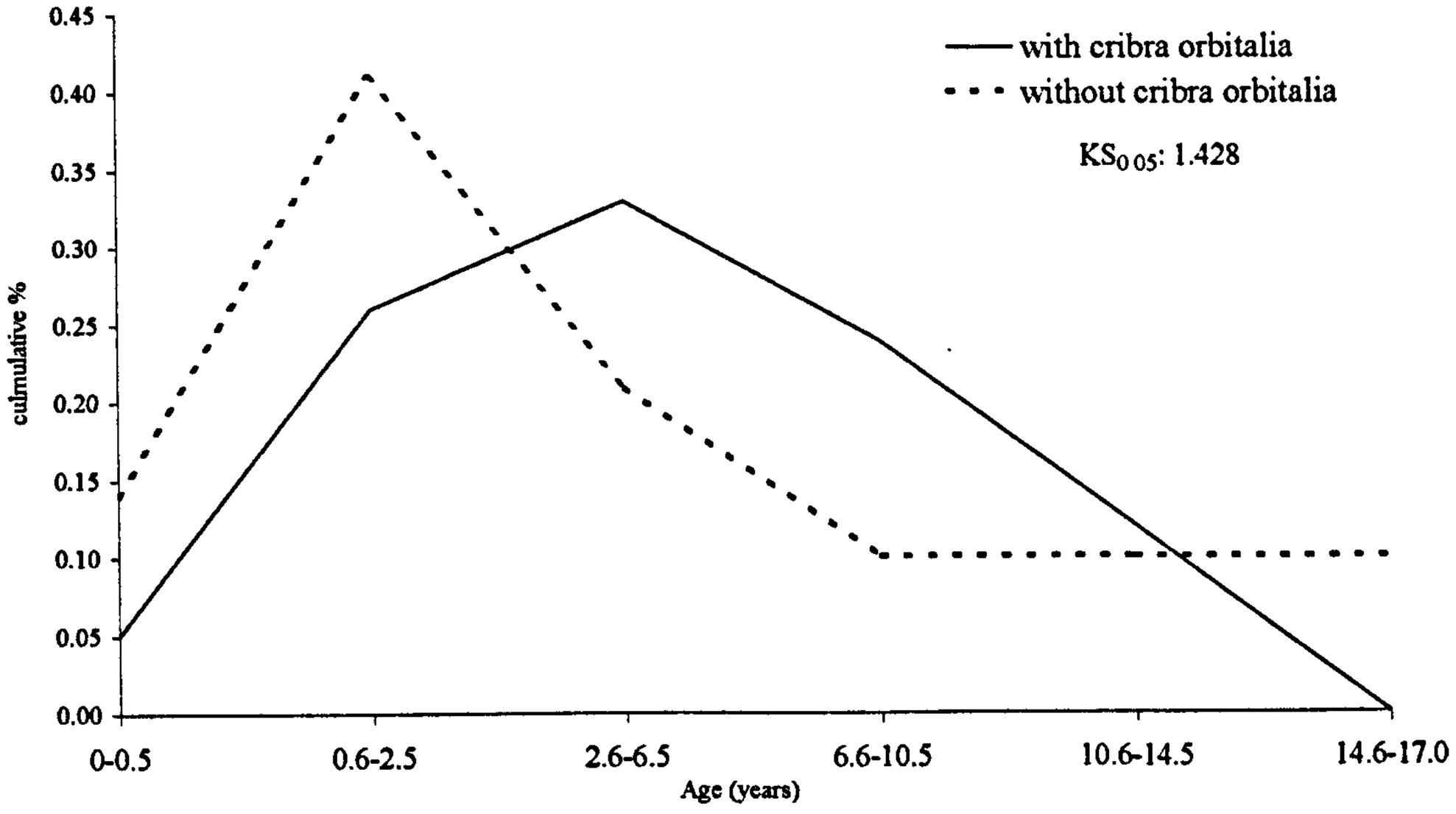


Figure 7.26 Mortality curves of individuals with and without cribra orbitalia

Wharram Percy

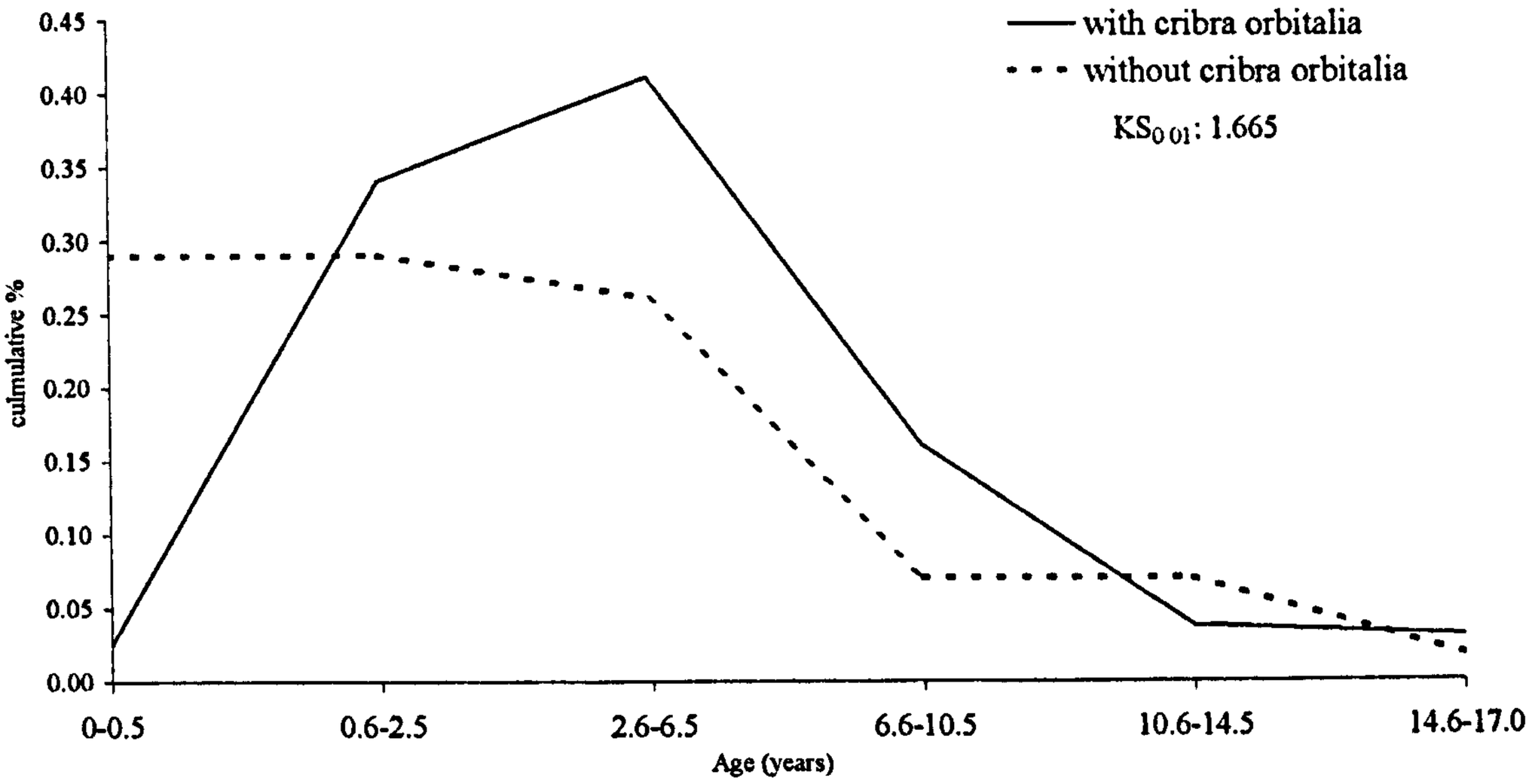


Figure 7.27 Mortality curves of individuals with and without endocranial lesions
Raunds Furnells

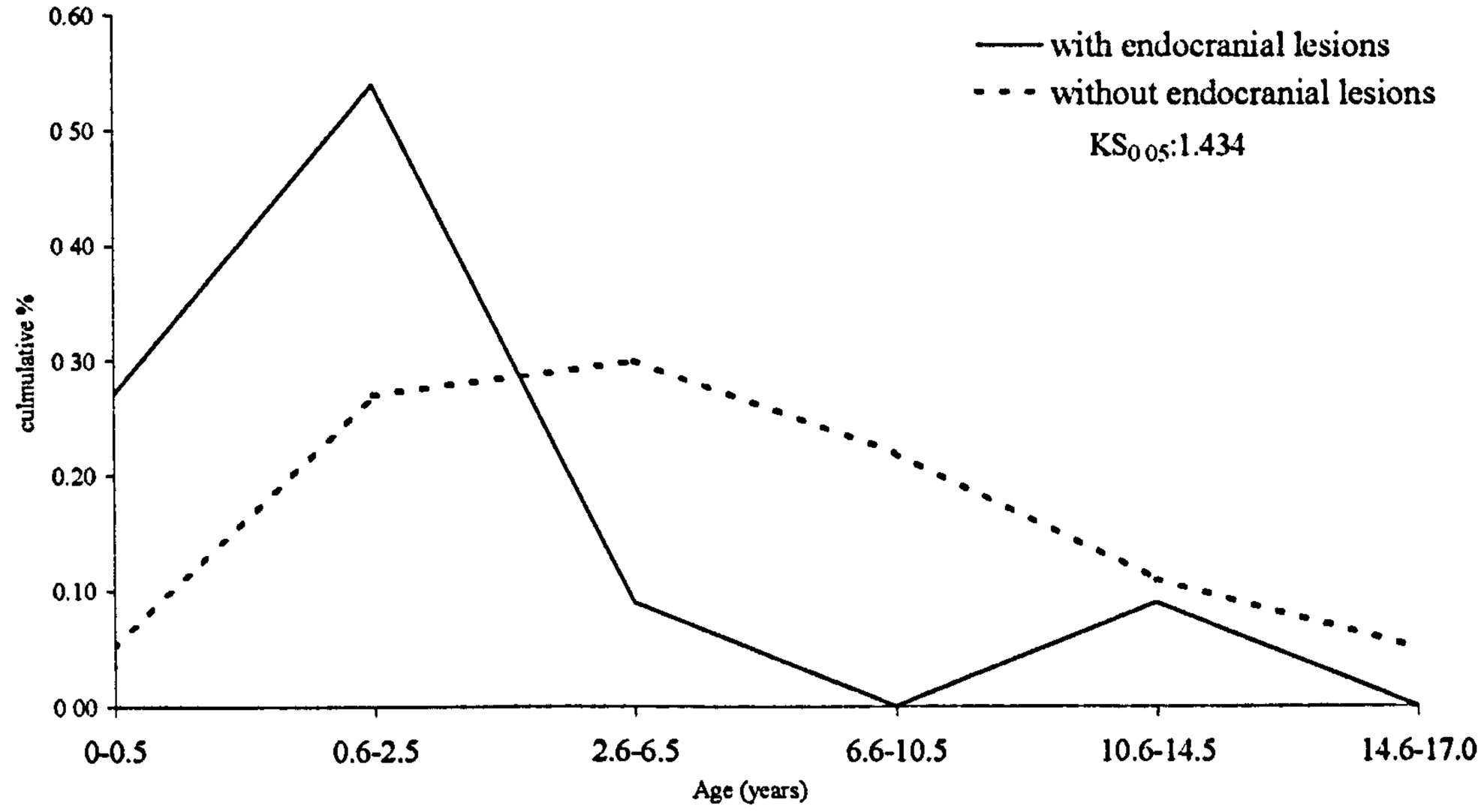
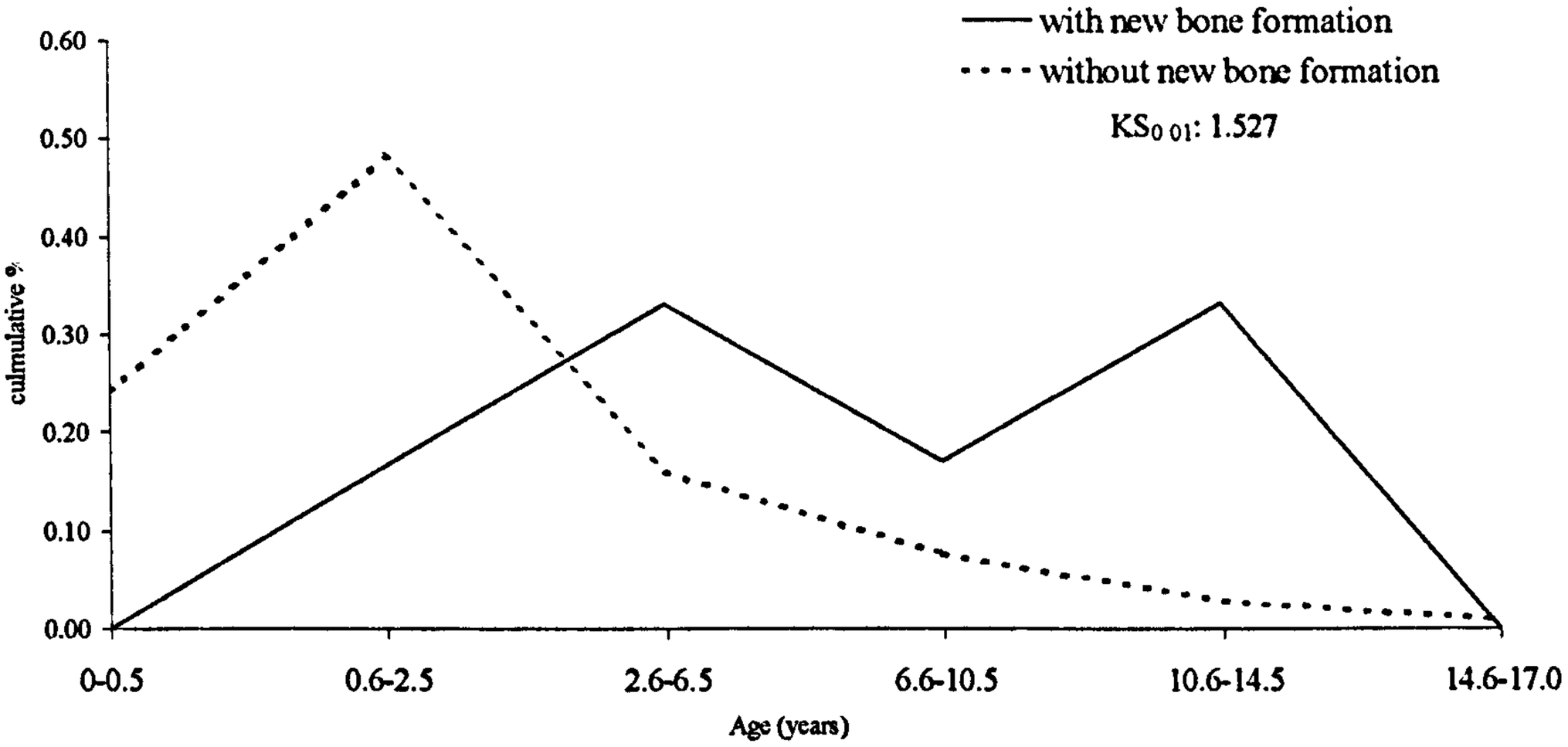


Figure 7.28 Mortality curves of individuals with and without new bone formation
Christ Church Spitalfields



7.5.4 Association between Stress Indicators

In previous studies, Palkovich (1987) and Grauer (1993) have found positive relationship between cribra orbitalia and periostitis and others have tested the association between Harris lines and enamel hypoplasias (McHenry and Schultz, 1976; Clarke, 1982; Maat, 1984; Mays, 1995). Table 7.9 shows the number and percentage of individuals with two or more indicators of stress in each site. Any relationship between stress indicators was tested using the Yule's Q measure of association. This statistic measures both the positive and negative covariation between the stress indicators where: 1.0 indicates a perfect negative covariation, +1.0 a perfect positive covariation and 0.0 indicates no association (Shennan, 1997). In order to ensure large sample sizes, individuals with multiple indicators of stress were combined from all samples and it was assumed that the aetiology of the lesions did not change between the sites. Table 7.10 presents the results of this test on the lesions most commonly found to be associated and numbers in bold show a near positive relationship.

Table 7.10 Test of association between stress indicators

Indicators of Stress	*Q*
1. Cribra orbitalia and enamel hypoplasias	-0.36
2. Cribra orbitalia and Harris lines	0.08
3. Cribra orbitalia and non-specific infections	**0.46**
4. Cribra orbitalia and endocranial lesions	-0.29
5. Enamel hypoplasias and Harris lines	0.07
6. Enamel hypoplasias and dental disease	**0.55**
7. Enamel hypoplasias and bone infections	0.28
8. Harris lines and bone infections	0.17
9. Maxillary sinusitis and dental disease	**0.45**

Despite the high percentages of individuals showing two or more lesions (Table 7.9), none of the indicators of stress proved to be perfectly associated, neither negatively nor positively. However, if values between 0.5 or –0.5 are taken as indicative of a trend, then cribra orbitalia and non-specific infections such as periostitis appear to be positively associated (3). Dental disease also shows a trend to be associated with enamel hypoplasias (6) and maxillary sinusitis (9).

Table 7.9 Association between stress indicators

Cribra orbitalia	*Hypoplasias*		*Endocranial lesions*		*New bone formation*		*Harris lines*		*Maxillary sinusitis*	
Site	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%
Raunds Furnells	12 (35)	34	3 (46)	6.5	6 (40)	15	19 (26)	73	1 (17)	6
St. Helen	18 (50)	36	3 (58)	5	7 (28)	25	12 (19)	63	5 (33)	15
Wharram Percy	24 (79)	30	16 (115)	14	10 (64)	16	21 (65)	32	6 (54)	11
Spitalfields	13 (47)	28	2 (54)	4	5 (59)	8	5 (13)	38	1 (20)	5
Total	67 (211)	32	24 (273)	9	28 (191)	15	57 (123)	46	13 (124)	10

Enamel hypoplasias	*Cribra orbitalia*		*Endocranial lesions*		*New bone formation*		*Harris lines*		*Maxillary sinusitis*	
Site	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%
Raunds Furnells	12 (20)	60	1 (22)	4.5	6 (19)	31.5	8 (13)	61.5	2 (11)	18
St. Helen	18 (22)	82	2 (25)	8	4 (22)	18	10 (15)	67	3 (19)	16
Wharram Percy	24 (33)	73	4 (35)	11	3 (18)	17	8 (20)	40	2 (29)	7
Spitalfields	13 (20)	65	1 (16)	6	2 (19)	10.5	3 (3)	100	1 (10)	10
Total	67 (95)	70.5	8 (98)	8	15 (78)	19	29 (51)	57	8 (69)	11.5

New bone formation	*Cribra orbitalia*		*Endocranial lesions*		*Hypoplasias*		*Harris lines*		*Maxillary sinusitis*	
Site	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%
Raunds Furnells	6 (13)	46	1 (15)	7	6 (12)	50	3 (8)	37.5	1 (7)	14
St. Helen	7 (8)	87.5	1 (10)	10	4 (10)	40	8 (10)	80	2 (5)	40
Wharram Percy	10 (16)	62.5	4 (18)	22	3 (11)	27	7 (17)	41	1 (8)	12.5
Spitalfields	5 (5)	100	1 (4)	25	2 (4)	50	1 (1)	100	1 (3)	33
Total	28 (42)	67	7 (47)	15	15 (37)	40.5	19 (36)	53	5 (23)	22

Endocranial lesions	*Cribra orbitalia*		*New bone formation*		*Hypoplasias*		*Harris lines*		*Maxillary sinusitis*	
Site	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%
Raunds Furnells	3 (10)	30	1 (9)	11	1 (7)	14	3 (8)	37.5	0 (4)	0
St. Helen	3 (9)	33	1 (7)	14	2 (8)	25	1 (3)	33	0 (4)	0
Wharram Percy	16 (30)	53	14 (28)	50	4 (14)	28.5	5 (10)	50	0 (8)	0
Spitalfields	2 (5)	40	1 (5)	20	1 (4)	25	0 (1)	0	0 (1)	0
Total	24 (54)	44	17 (49)	35	8 (33)	24	9 (22)	41	0 (17)	0

Maxillary sinusitis	*Cribra orbitalia*		*New bone formation*		*Hypoplasias*		*Harris lines*		*Endocranial Lesions*	
Site	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%
Raunds Furnells	1 (2)	50	1 (2)	50	2 (2)	100	0 (1)	0	0 (2)	0
St. Helen	5 (6)	83	2 (4)	50	3 (5)	60	1 (2)	50	0 (6)	0
Wharram Percy	6 (8)	75	1 (5)	20	2 (8)	25	2 (6)	33	0 (8)	0
Spitalfields	1 (2)	50	1 (2)	50	1 (2)	50	0 (0)	0	0 (2)	0
Total	13 (18)	72	5 (13)	38	8 (17)	47	3 (9)	33	0 (18)	0

Harris lines	*Cribra orbitalia*		*New bone formation*		*Hypoplasias*		*Maxillary sinusitis*		*Endocranial Lesions*	
Site*	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%	N1 (N2)	%
Raunds Furnells	19 (26)	73	3 (28)	11	8 (26)	31	0 (14)	0	3 (28)	11
St. Helen	12 (16)	75	8 (38)	21	10 (22)	45	1 (13)	8	1 (19)	5
Wharram Percy	21 (36)	58	7 (45)	15.5	8 (30)	27	2 (25)	8	5 (42)	12
Total	52 (78)	67	18 (111)	16	26 (78)	33	3 (52)	6	9 (89)	10

	Hypoplasias and dental disease		*Sinusitis and dental disease*	
Site	N1 (N2)	%	N1 (N2)	%
Raunds Furnells	3 (23)	13	0 (2)	0
St. Helen	14 (30)	47	2 (8)	25
Wharram Percy	5 (39)	13	2 (6)	33
Spitalfields	10 (22)	45	1 (2)	50
Total	32 (114)	28	5 (18)	28

N1= number of individuals with both lesions; N2= number observed
*%=percentage of individuals with both lesions; *data not complete for Christ Church Spitalfields*

7.5.5 CRIBRA ORBITALIA

The prevalence of cribra orbitalia between sites and within age groups was not statistically significant. However, when examined in more detail, at Spitalfields there was a peak in the frequency of cribra orbitalia in the 0.6-2.5 year age category, which is earlier than at the other sites, where the orbital lesions occurred most frequently in the 2.6-6.5 year age category (Figure 7.29). Although the peak was not significant within the site (0-0.5-0.6-2.5 = X^2=2.73) or between sites (X^2=0.17).

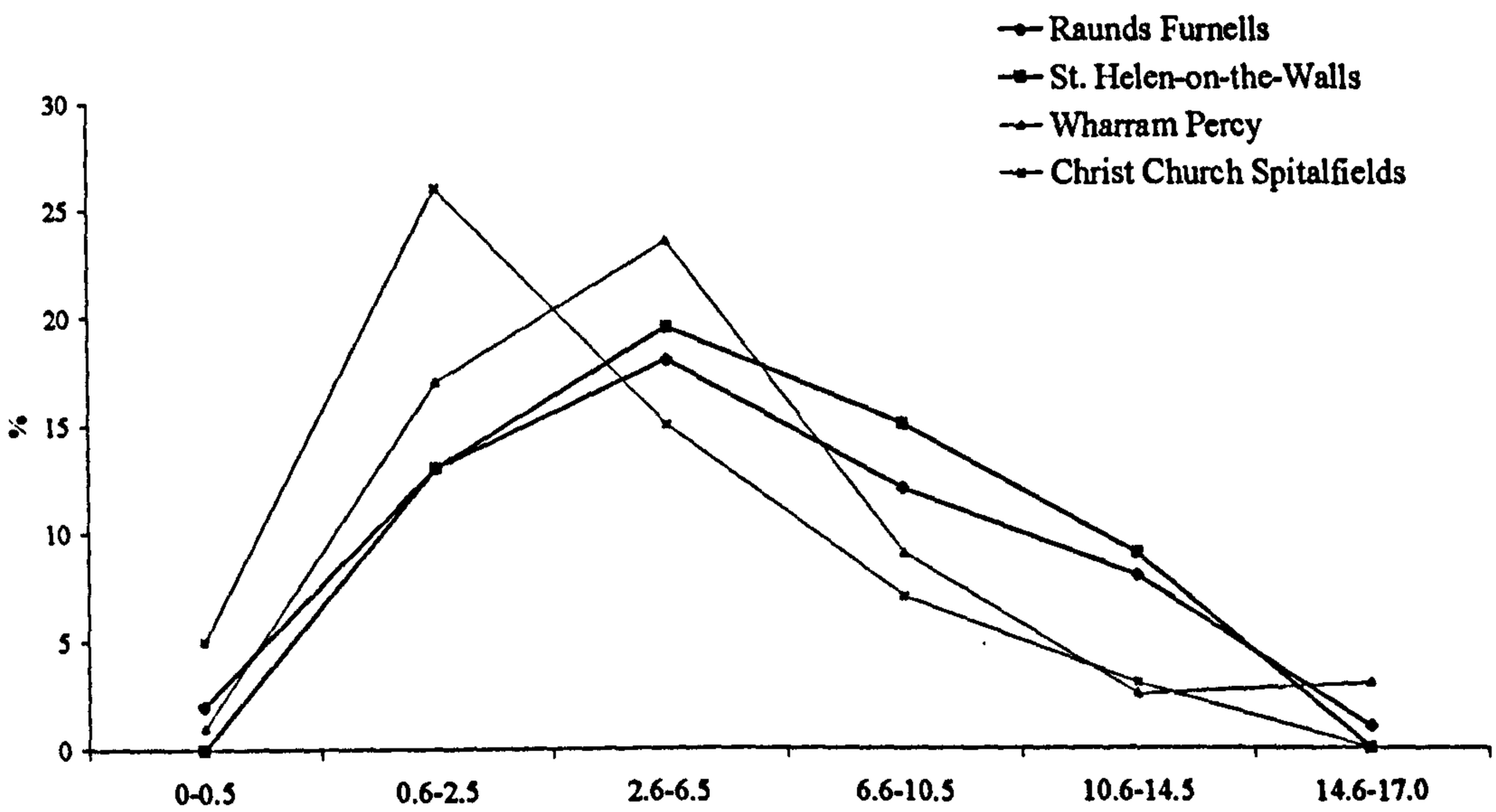


Figure 7.29 Frequency of cribra orbitalia

The severity of cribra orbitalia between the sites showed that most of the individuals displayed lesions corresponding to Stuart-Macadam's Grade 2 (1991), scattered fine foramina (Figure 7.30) and that Wharram Percy had significantly more of these lesions than Raunds Furnells (X^2=6.81; P=0.001; d.f=1). The more severe form of the lesion (Grade 3) was significantly more common in the urban samples (SHvRF: X^2=4.0; SHvWP: X^2=4.29: CSvRF: X^2=5.11; CSvWP: X^2=5.77; P=0.05; d.f=1).

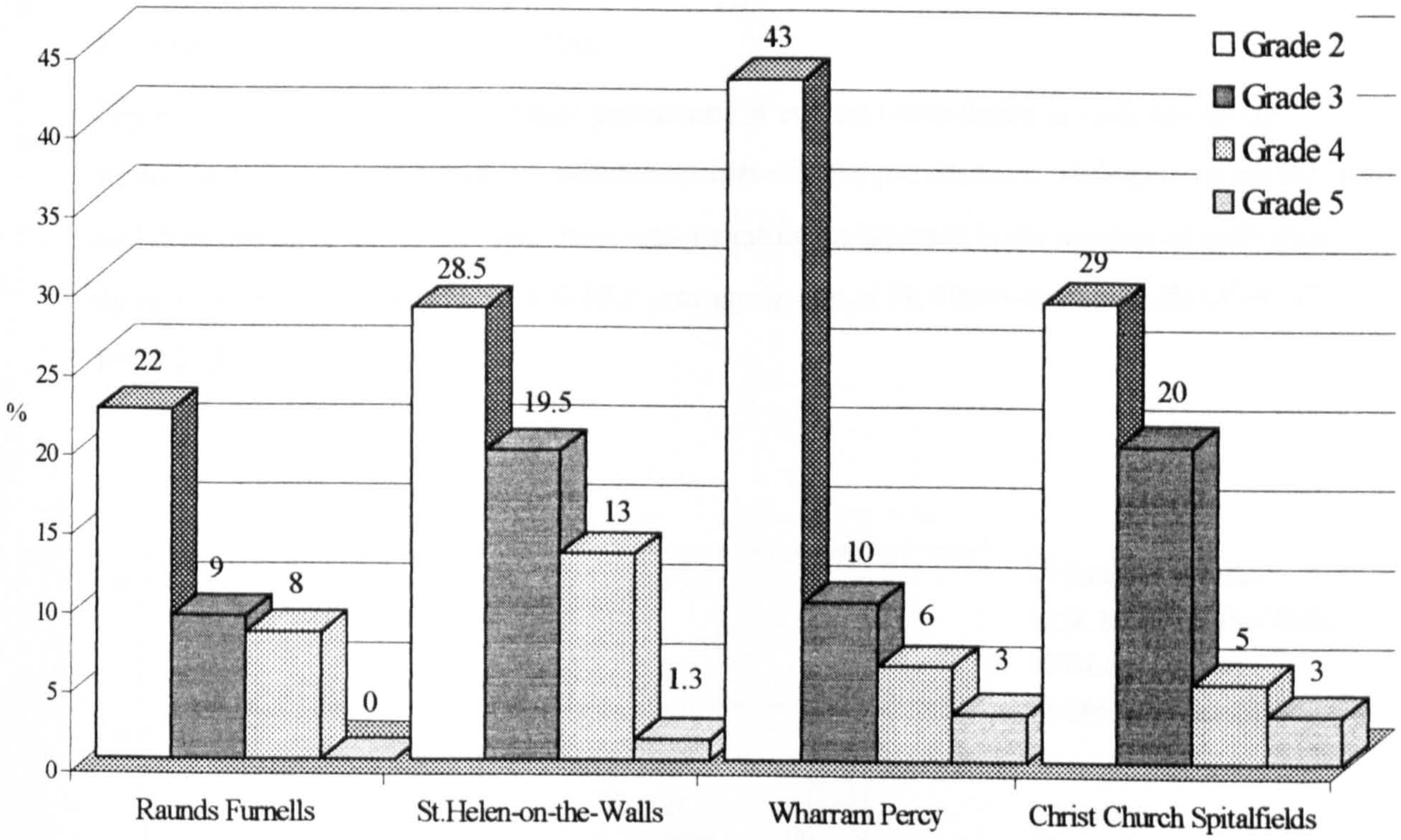


Figure 7.30 Severity of cribra orbitalia

Figure 7.31 shows the percentage of healed and active lesions, and in all of the sites active lesions were more common than healed lesions. Numbers were too small to test the prevalence of healed and active lesions within age groups. However, at Raunds Furnells healed lesions did not occur until the 2.6-6.5 year age group compared to 0.6-2.5 years at the other sites. Hence, although Raunds Furnells did not have the highest prevalence or the severest form of lesions, the cause of cribra orbitalia appears to have persisted for longer in the children from this Anglo-Saxon village.

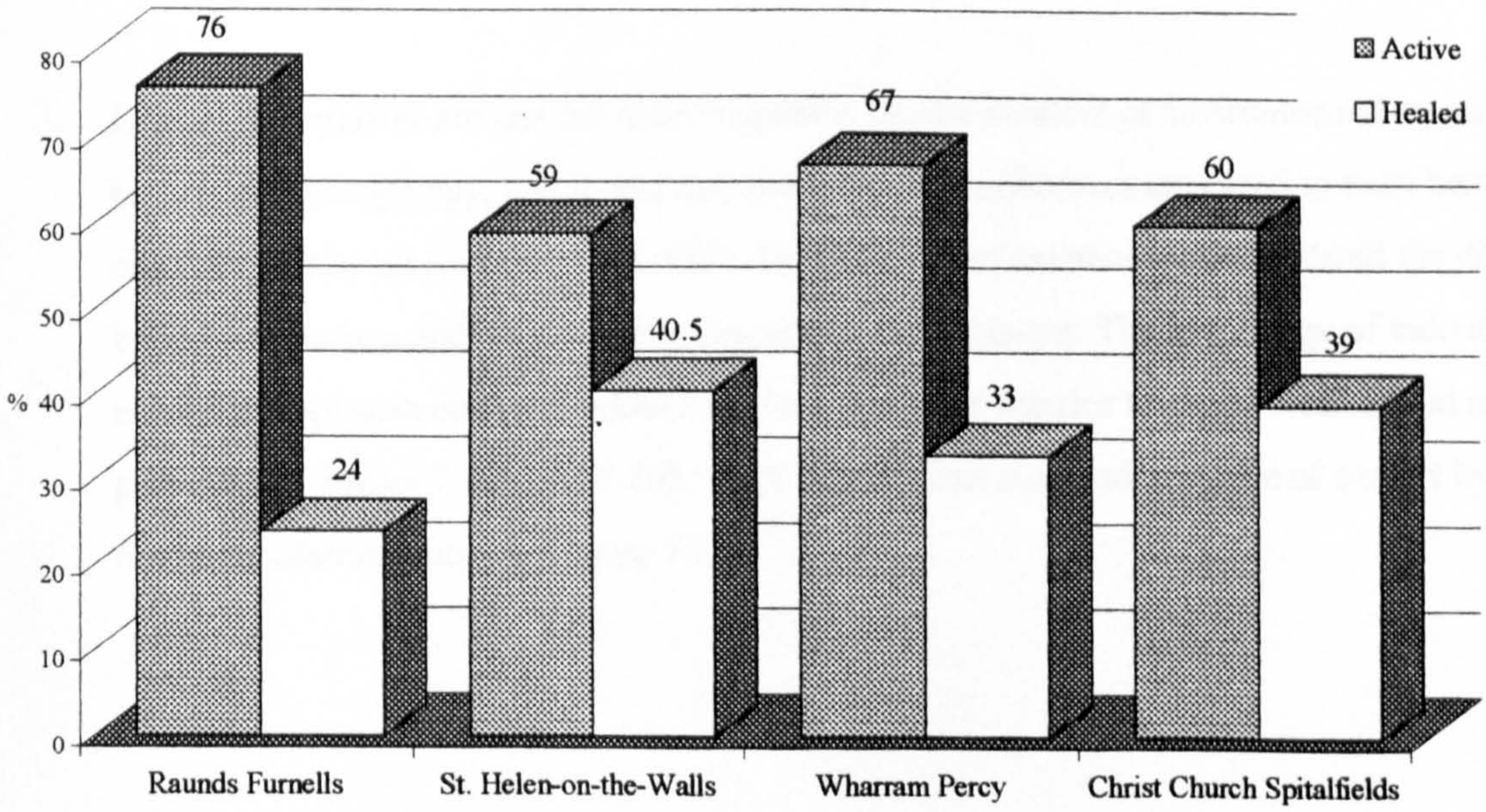


Figure 7.31 Percentage of healed and active lesions

7.5.6 DENTAL ENAMEL HYPOPLASIAS

Figure 7.32 illustrates the percentage prevalence of enamel hypoplasias in each age group. Although there were no significant differences between the prevalence of defects between the sites and their comparative age groups, there was a significant increase in the number of individuals dying with enamel defects in the 6.6-10.5 year age group at St. Helen-on-the-Walls (X^2=6.47; P=0.01; d.f.=1).

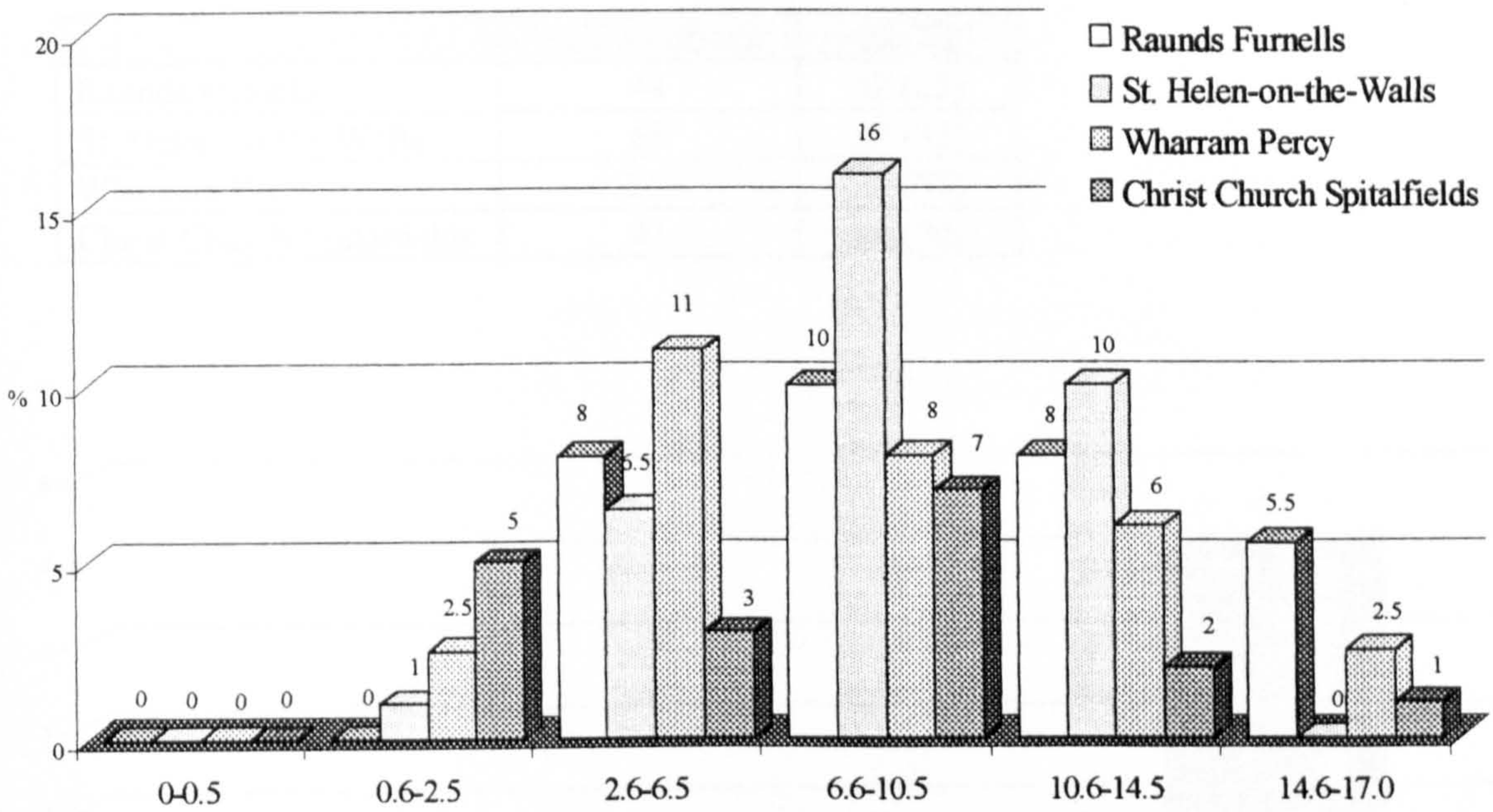


Figure 7.32 Percentage prevalence of enamel hypoplasias in each age group.

Enamel hypoplasias are one the most frequently used indicators of environmental stress in biological anthropology, and it was surprising that no differences appeared to exist between these environmentally diverse sites. But when the defects were examined in more detail the difference between the urban and rural samples becomes more apparent. The percentage of individuals with enamel hypoplasias on the deciduous and/or permanent anterior teeth were calculated and are presented in Tables 7.10a and 7.10b. Both of the urban sites had evidence of enamel hypoplasias on the deciduous dentition (Figure 7.33).

Table 7.10a Prevalence of hypoplasias on the deciduous dentition

	with dentitions	% (N)
Raunds Furnells	38	0 (0)
St. Helen-on-the-Walls	52	2 (1)
Wharram Percy	93	0 (0)
Christ Church Spitalfields	68	7 (5)

Table 7.10b Prevalence of hypoplasias on the permanent dentition

	with dentitions	% (N)
Raunds Furnells	44	52 (23)
St. Helen-on-the-Walls	65	49 (32)
Wharram Percy	121	30 (37)
Christ Church Spitalfields	41	49 (20)

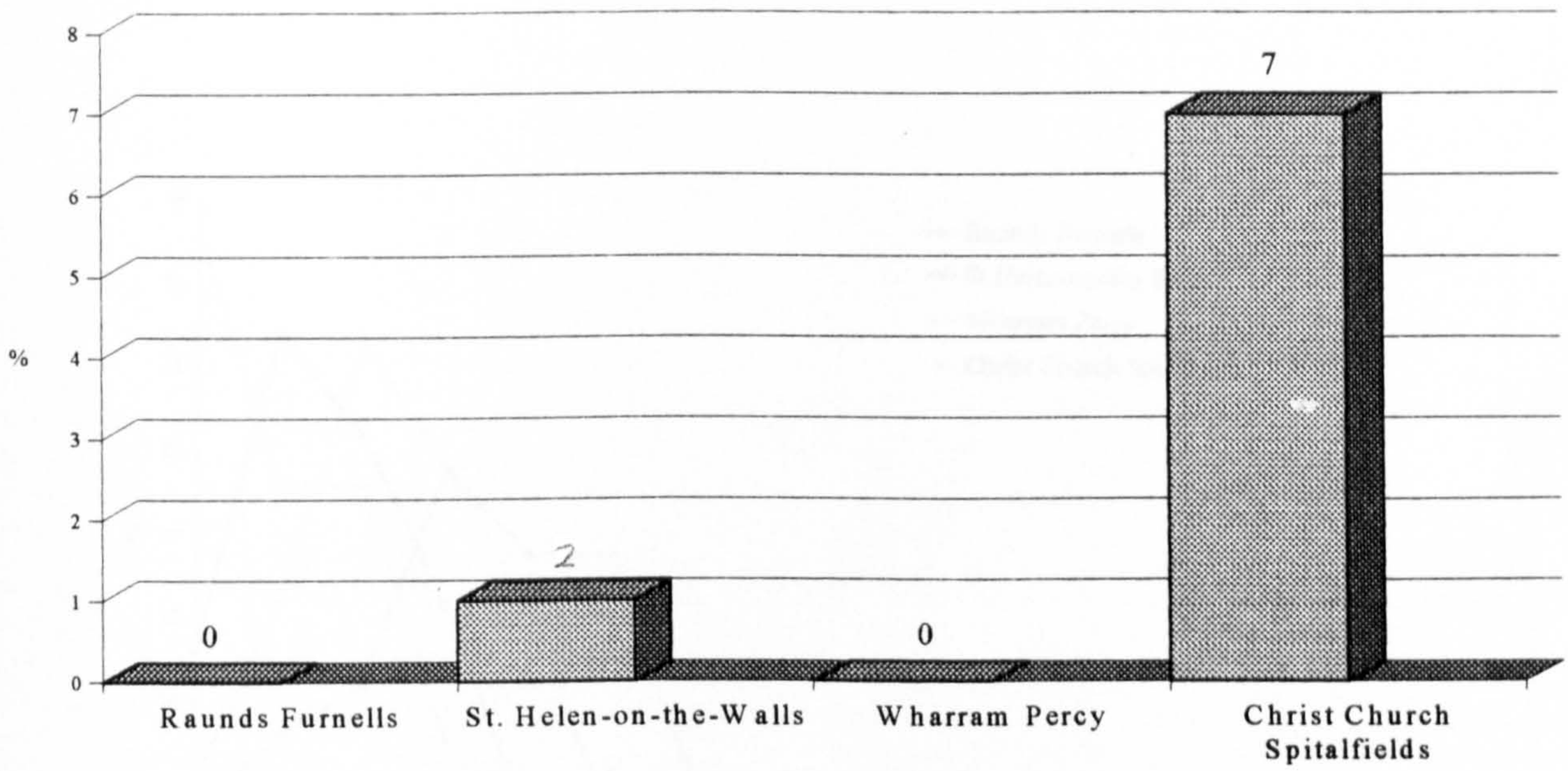


Figure 7.33 Prevalence of deciduous enamel hypoplasias

Once the enamel hypoplasias had been divided into 'deciduous' and 'permanent' teeth, the prevalence of enamel hypoplasias between the sites was re-tested. This time, the later medieval sites of Wharram Percy and St. Helen had significantly higher prevalences of enamel hypoplasias ($X^2 = 9.73$, P=0.025, d.f.=3) than either Raunds Furnells or Spitalfields.

The ages at which the enamel hypoplasias were formed were calculated using the method outlined by Goodman and Rose (1991). The ages of formation for each tooth are presented in Appendix IX and Table 7.11, and Figure 7.34 summarise the results. All the sites showed a peak in hypoplasia formation on the permanent dentition at different ages (Table 7.1).

Table 7.11 Peak age of enamel hypoplasia formation

Site	*Age (years)*
Raunds Furnells	2.00
St. Helen-on-the-Walls	1.00
Wharram Percy	1.5
Christ Church Spitalfields	0.5

If the position of the defect on the crown of a tooth is a reliable indicator of the timing of the stress episode that caused the defect, then all the sites show different ages as being times of the most severe stress. Interestingly, as the dates of the sites increase, the ages of peak stress occur earlier, with Raunds Furnells showing a peak in hypoplasias at two years and Spitalfields at six months.

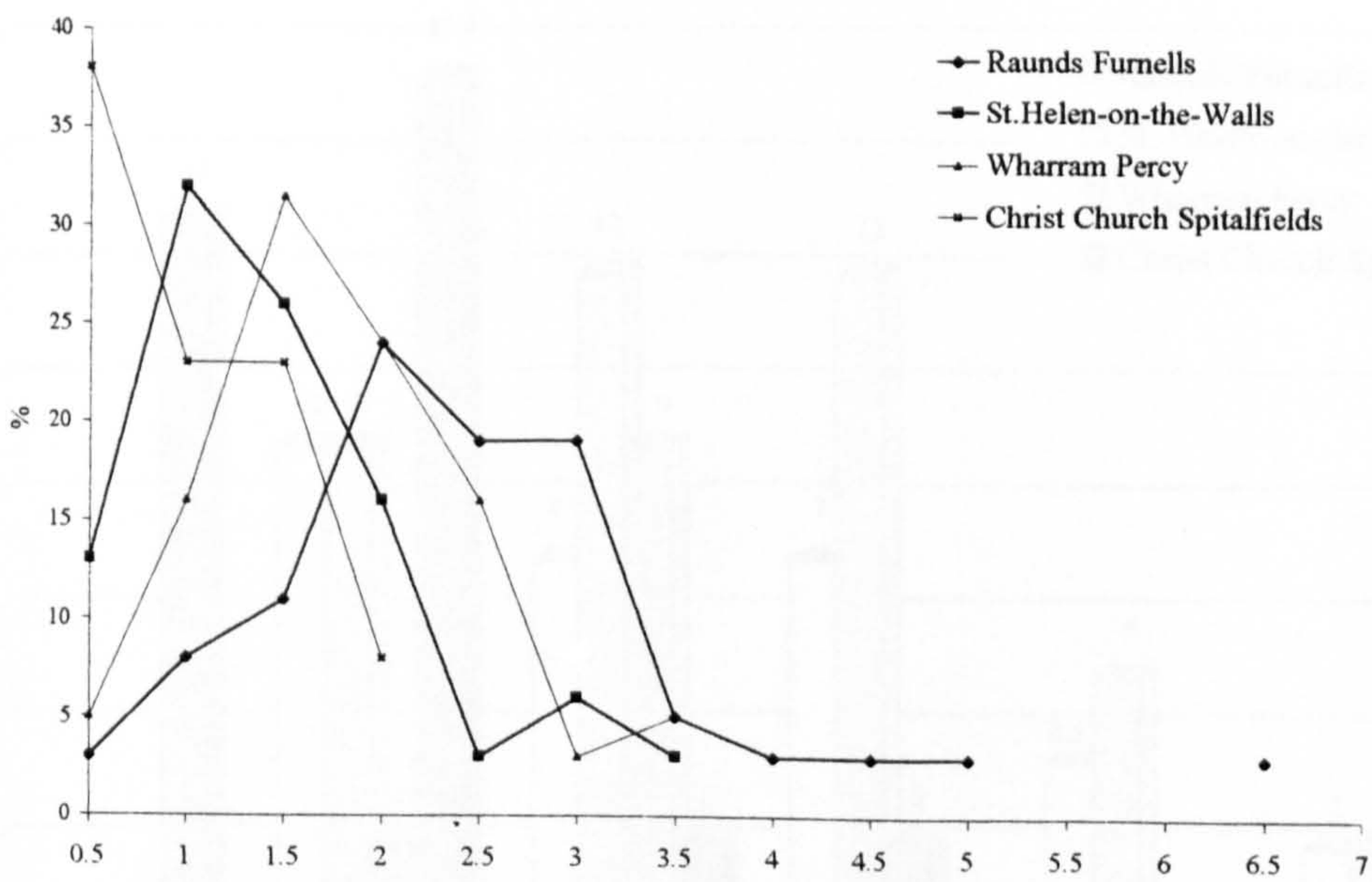


Figure 7.34 Frequency of enamel hypoplasia formation (half year age categories)

7.5.7 HARRIS LINES

Figure 7.16 shows that the prevalence of Harris lines (Grade 2) was significantly higher in the urban than the rural sites (X^2=20.06, P=0.001, d.f.=3). Due to the exposure of the Spitalfields bones to lead in the burial environment, and the lack of radiographs for the older age groups, Spitalfields was omitted from the study after the 2.6-6.5 year age category. At the other sites, the maximum number of lines occurring in any one individual was the same (5 lines) but the average number of lines differed. At Raunds Furnells there was an average of 2.4 lines, at St. Helen 2.1 lines and at Wharram Percy this number was smaller with the average number of lines per individual being 1.7.

When the prevalence of Harris lines in each age category was examined (Figure 7.35), there were significantly more lines in the children dying in the 6.6-10.5 year age category at Wharram Percy than at the other two sites (X^2=5.73, P=0.05, d.f.=2). However, the prevalence of Harris lines in the urban site of St. Helen continues to remain high throughout the age categories, even though it declines in Raunds Furnells after 0.6-2.5 years.

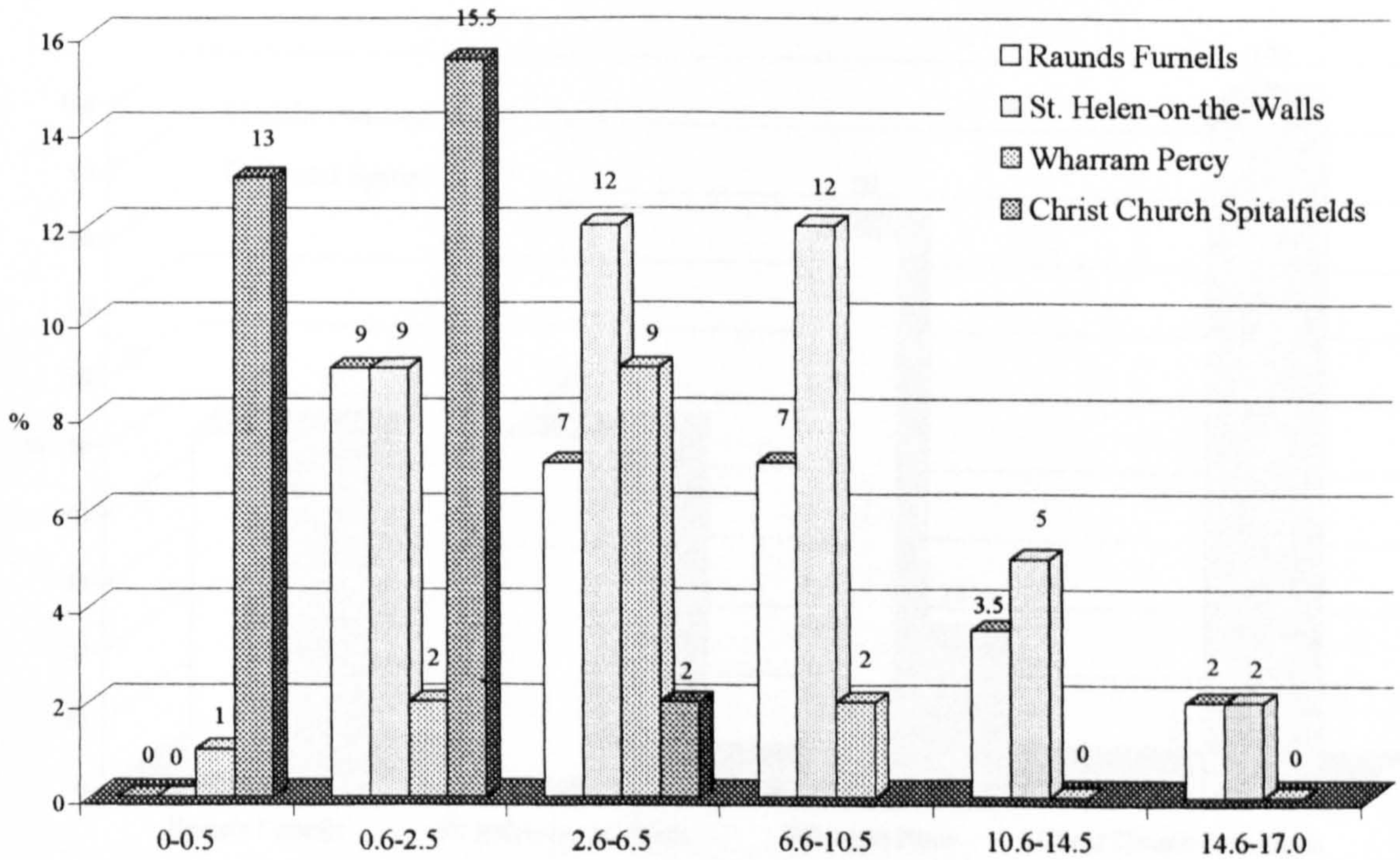


Figure 7.35 Percentage prevalence of Harris lines (Grade 2 only) in each age category

7.6 NON-SPECIFIC INFECTIONS

7.6.1 MAXILLARY SINUSITIS

Chronic maxillary sinusitis can be caused by a dental spread of infection or through sinus irritation as the result of air pollution or allergies. A previous study examining the prevalence of maxillary sinusitis in the adults from Wharram Percy and St. Helen revealed maxillary sinusitis to be more common in the urban site (Lewis *et al.*, 1995). When the prevalence of sinusitis was tested between the sites, there were no significant differences between the urban and rural non-adults (X^2=3.59, d.f. =3), although St. Helen had the highest prevalence (17%) (see Figure 7.16).

When the cases of maxillary sinusitis were divided into those with evidence of dental disease (caries and abscesses) and those without, both Raunds Furnells (2 cases) and St. Helen (6 cases) showed equal numbers of dental and non-specific sinusitis. At Wharram Percy (8 cases) most of the cases (6; 80%) had no evidence of dental disease suggesting that respiratory infections were probably the major aetiological factor and, at Spitalfields, the only individual with sinusitis had evidence of dental disease (Figure 7.36).

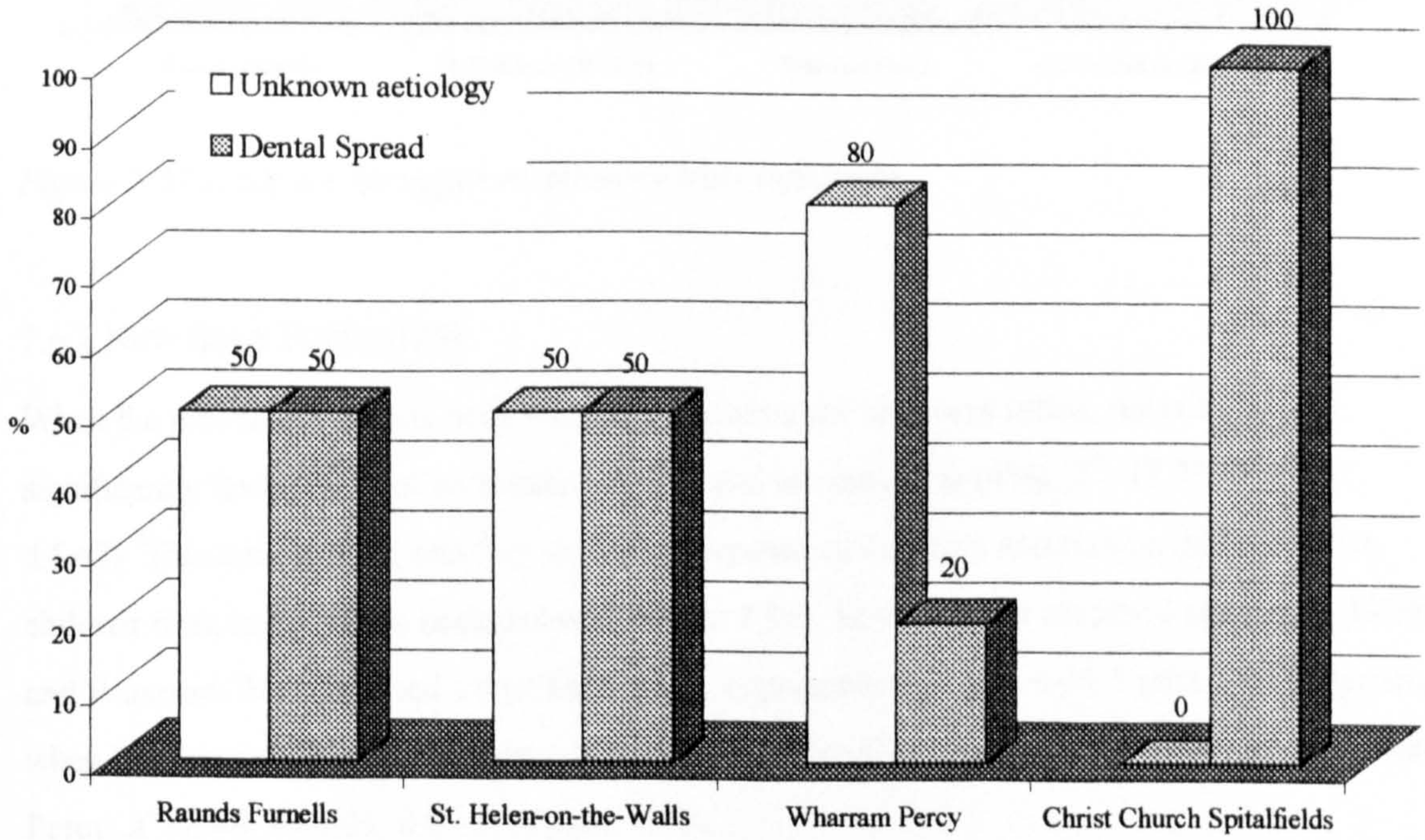


Figure 7.36 Aetiology of maxillary sinusitis

In order to examine the prevalence of respiratory disorders between the urban and rural samples further, other upper respiratory tract infections, denoted by evidence from pitting on the nasal surface of the palate (rhinitis) and rib periostitis was analysed (Figure 7.37, listed in Appendix X).

Once again, rural sites of Wharram Percy and Raunds Furnells showed the highest prevalence of respiratory infections.

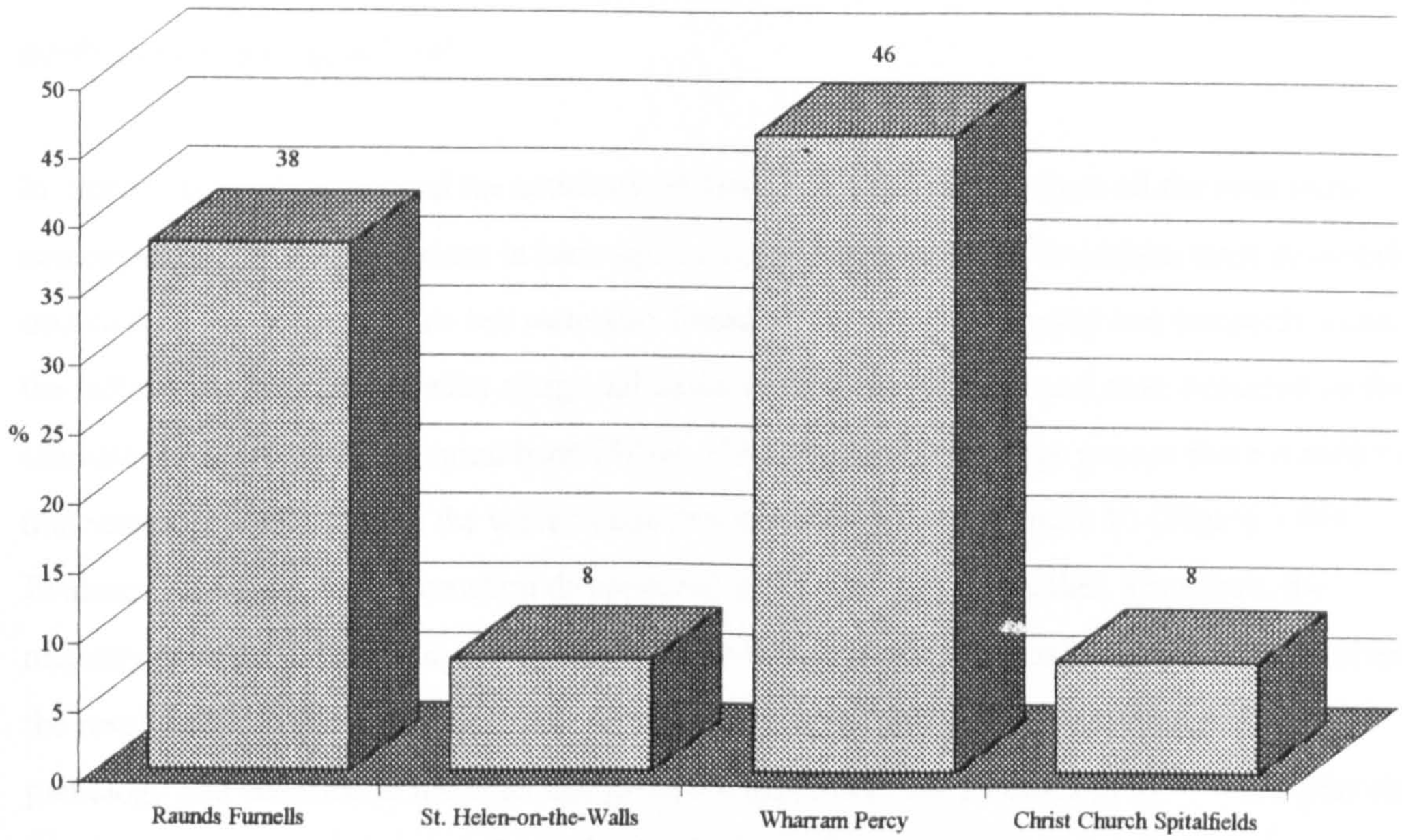


Figure 7.37 Evidence for upper respiratory tract infections

7.6.2 New Bone Formation

When the prevalence of new bone formation between the sites was tested, Spitalfields had significantly fewer cases of periostitis, osteitis and osteomyelitis (4%) (X^2=17.73, P=0.001, d.f.=3). This may suggest that they were less exposed to infection and trauma than the other children from lower status backgrounds (Figure 7.16). Both the later medieval sites of St. Helen and Wharram Percy showed a significant peak in periostitis in the 6.6-10.5 year age categories, when Raunds Furnells and Spitalfields showed a decline (St. Helen: X^2=5.17, P=0.05; Wharram Percy: X^2=8.40, P=0.01, d.f.=1) (Figure 7.38).

When the severity of lesions was assessed, the majority of lesions were localised patches of woven bone indicative of an active inflammatory process (Figure 7.39). Wharram Percy had significantly

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7.6.3 Endocranial Lesions

The majority of individuals displaying endocranial lesions were aged between 0.6-2.5 years of age (53%). However, four cases occurred in older individuals. At Raunds Furnells a child with a dental age of 10.7 was affected and at St. Helen a child around 12 years of age had endocranial lesions. At Wharram Percy, two children around nine years of age were affected. When the prevalence of endocranial lesions between the sites was tested, Spitalfields had significantly lower levels than any of the other sites (X^2=9.0, P=0.05, d.f.=3), and Wharram Percy had the greatest number of cases (Figure 7.40).

In order to try and understand the aetiology of these lesions, the cases from all the sites were combined and the type of lesions in each age category was assessed. The lesions most commonly occurred on the occipital bone but were also found on the parietals, frontal and temporal bones. In the individuals under six months of age, all cases were of woven bone and most occurred on the cruciate eminence of the occipital bone (82%). However, in the later age groups there is evidence that vessels were forming in the woven bone deposits (see (c) in Appendix V) (Figure 7.41). Evidence for woven bone formation disappeared in the later age categories. Therefore, the majority of cases in the youngest age category (0-0.5 years) were probably non-pathological and the result of rapid growth in that area. A more widespread deposit is probably indicative of pathology and occurred in the older age groups with 66% of the 24 cases in the 0.6-2.5 year age category also having lesions on the parietals, frontal and temporal bones.

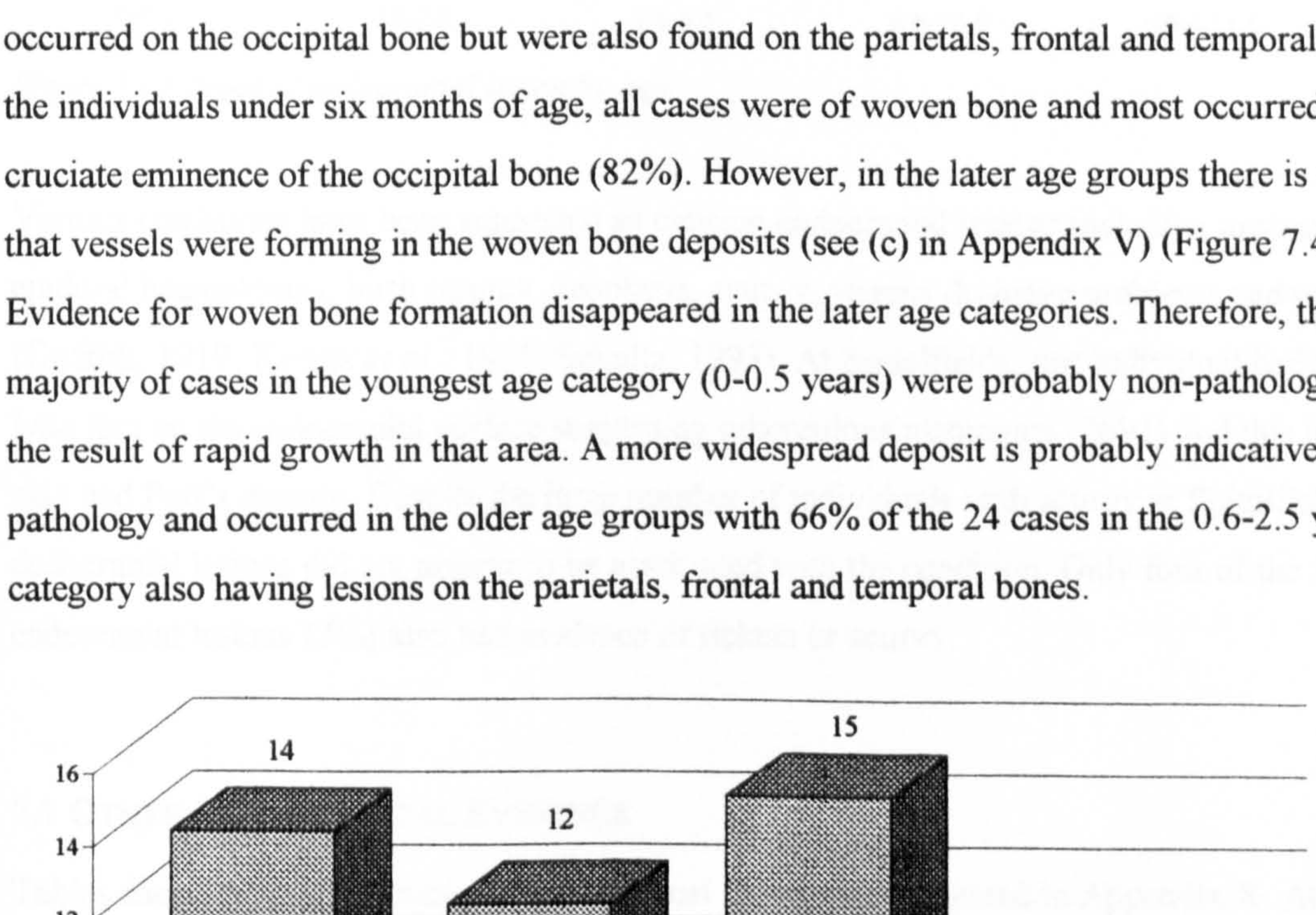

Figure 7.40 Prevalence of endocranial lesions

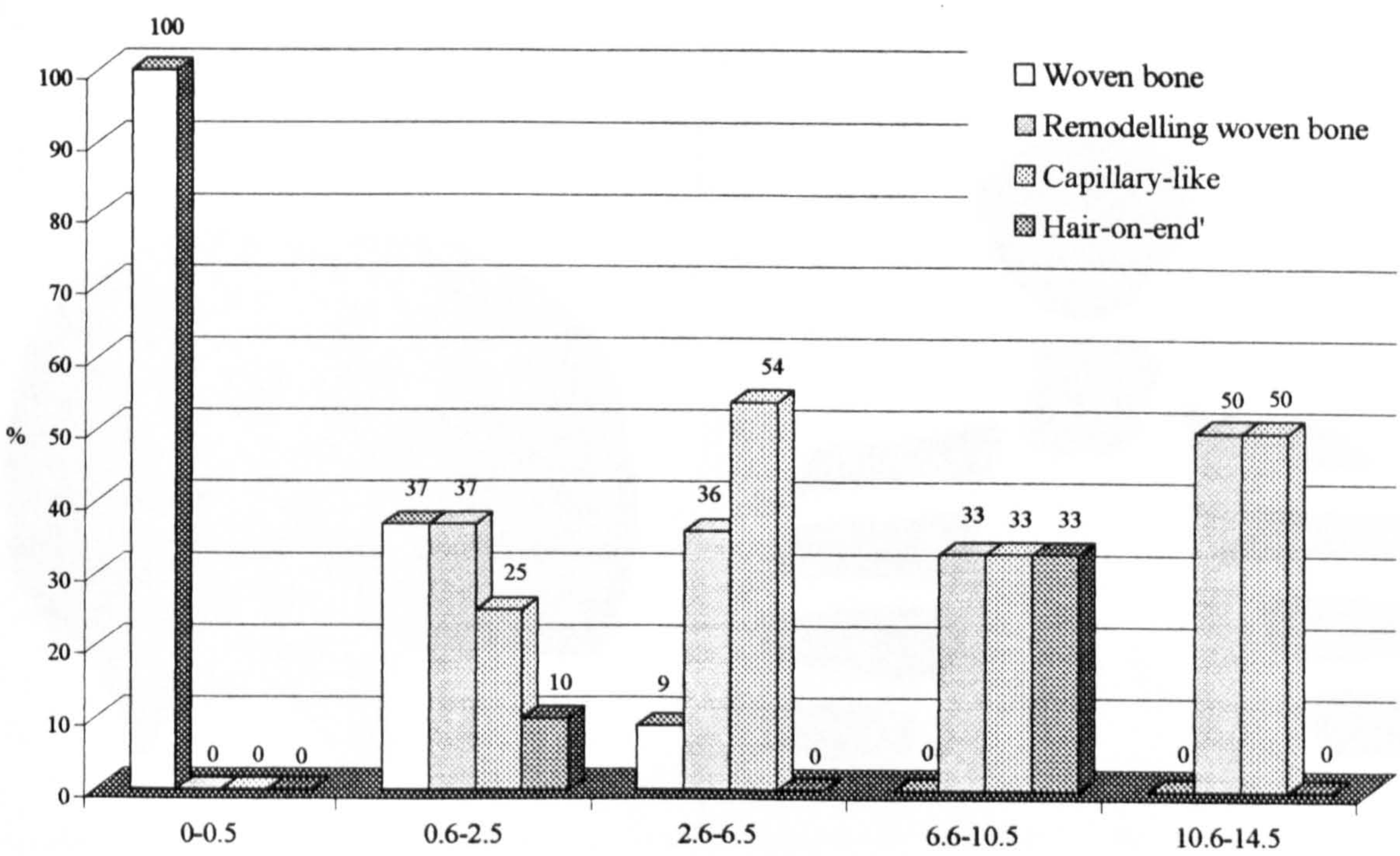

Figure 7.41 Types of endocranial lesion by age

Various conditions have been suggested as causing endocranial lesions including meningitis, epidural haematomas, birth trauma, neoplasia, scurvy, venous drainage problems and tuberculosis (Griffith, 1919; Kreutz *et al.*, 1995; Schultz, 1993). At Spitalfields, one individual had isolated lytic foci on the endocranial surface suggesting tuberculous meningitis (2691) and this individual also had Pott's disease. Despite the large number of individuals with scurvy at Spitalfields, endocranial lesions did not appear to be associated with the condition. Only four of the 57 cases of endocranial lesions (7%) also had evidence of rickets or scurvy.

7.7 Other Pathological Evidence

Tables showing the distribution of pathological lesions are presented in Appendix X. At Spitalfields there was evidence for medical investigation on the children's skeletons, with three skeletons showing evidence of an autopsy. A peri-mortem break was evident on the mid-shaft of a left humerus (2374) of one child and the frontal bone had been sectioned in two cases (2412 and 2455). A further skeleton (2235) had ribs cut to provide access to the thorax. Both cases of frontal bone autopsy were aged between 0.6-2.5 years but there were no pathological lesions (for example endocranial lesions) on the skeletons to suggest the reason for this procedure (Figure 7.42a and 7.42b). There was one case of neoplasm on the mandible of a child (2.6-6.5 years of age) at Wharram Percy (NA135).

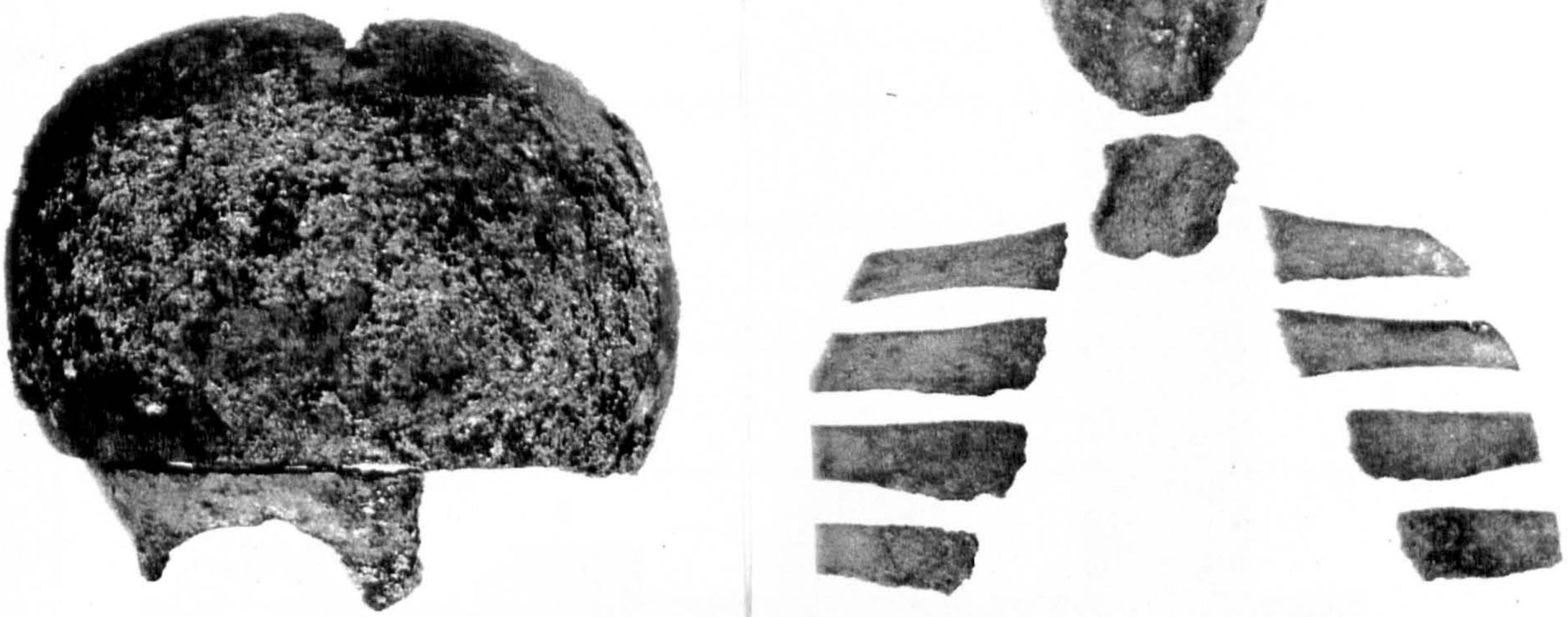

Figure 7.42a Sectioned frontal bone of a child from Spitalfields (2455); Figure 7.42b cut marks on the sternal ends of the ribs of a Spitalfields child (2235). From Molleson and Cox (1993).

7.7.1 Metabolic Disease

Rickets and scurvy were diagnosed using the criteria described by Ortner and colleagues in several recent papers (Ortner and Ericksen, 1997; Ortner and Mays, 1998; Ortner *et al.*, 1999), and Ortner and Mays (1998) and Molleson and Cox (1993) have previously published cases of metabolic disease at Wharram Percy and Spitalfields. The prevalence of metabolic disease at the four sites is presented in Figure 7.43 and descriptions of the lesions are contained in Appendix X. Due to the nature of the lesions, it was not always possible to distinguish between rickets and scurvy, particularly as both conditions commonly occurred together.

As metabolic disorders are commonly associated with retarded growth, it was expected that individuals from Spitalfields, with evidence for rickets and scurvy would be smaller than their peers. Therefore, scatter plots of individuals with and without metabolic disease were plotted up to two years of age and the distributions tested using the Kolmogorov-Smirnov statistic (Figure 7.44) Individuals with metabolic disease were significantly shorter than their peers.

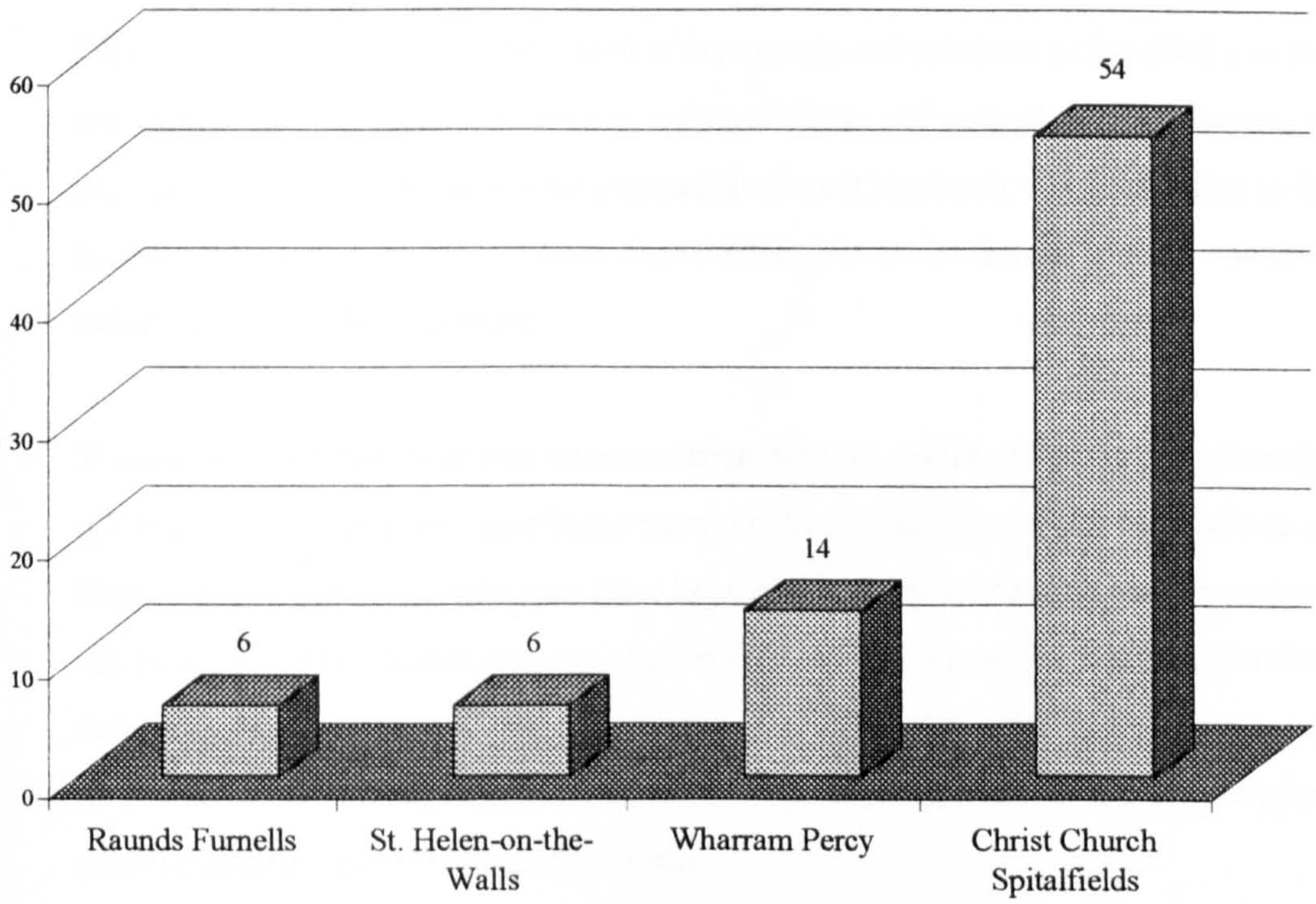

Figure 7.43 Prevalence of metabolic disease

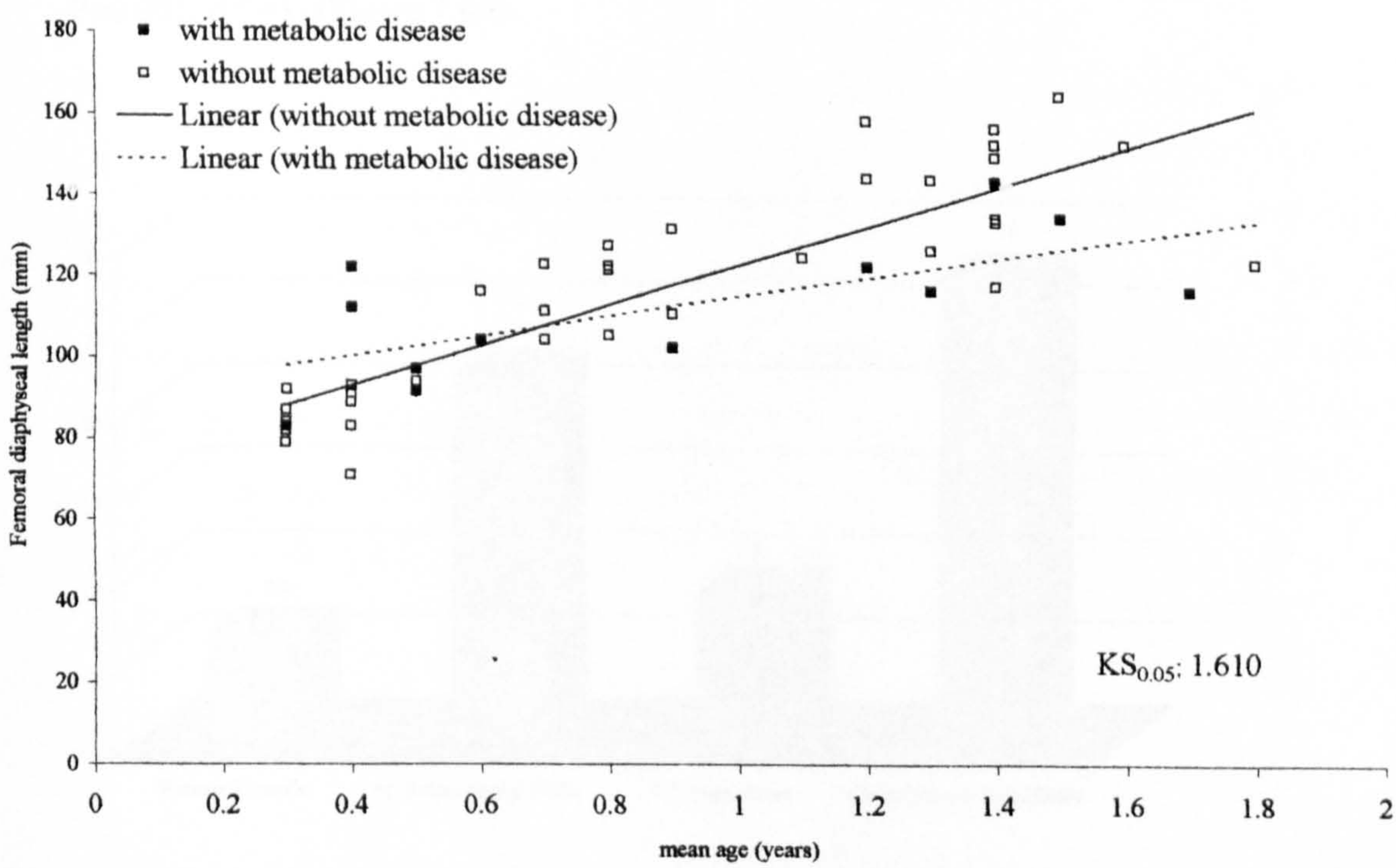

Figure 7.44 Growth profiles of individuals with and without metabolic disease at Christ Church Spitalfields.

Rickets and scurvy are also associated with anaemia and infection in the child and therefore, the association between metabolic disease, cribra orbitalia and enamel hypoplasias was tested using the Yule's Q statistic. Evidence for periostitis was not used as it was impossible to differentiate between the layers of immature bone deposited as part of the disease process and new bone indicating a secondary infection.

The test revealed that there was no association between cribra orbitalia and metabolic disease (Q=0.23) but that enamel hypoplasias were positively associated with the condition (Q=0.5). Some cases of cribra orbitalia may have been obscured by subsequent new bone deposition in the orbits as part of the disease process of scurvy. However, it is surprising that, if indicative of iron deficiency anaemia, the children with obvious signs of malnutrition are not displaying the lesion more frequently. The link between enamel hypoplasias and malnutrition is supported by its positive association with metabolic disease.

7.7.2 Dental Disease

The urban sites of St. Helen and Spitalfields had significantly higher rates of dental caries and abscesses on the deciduous and permanent molars compared to the rural children (X^2=25.23, P=0.001, d.f.=3) (Figure 7.45).

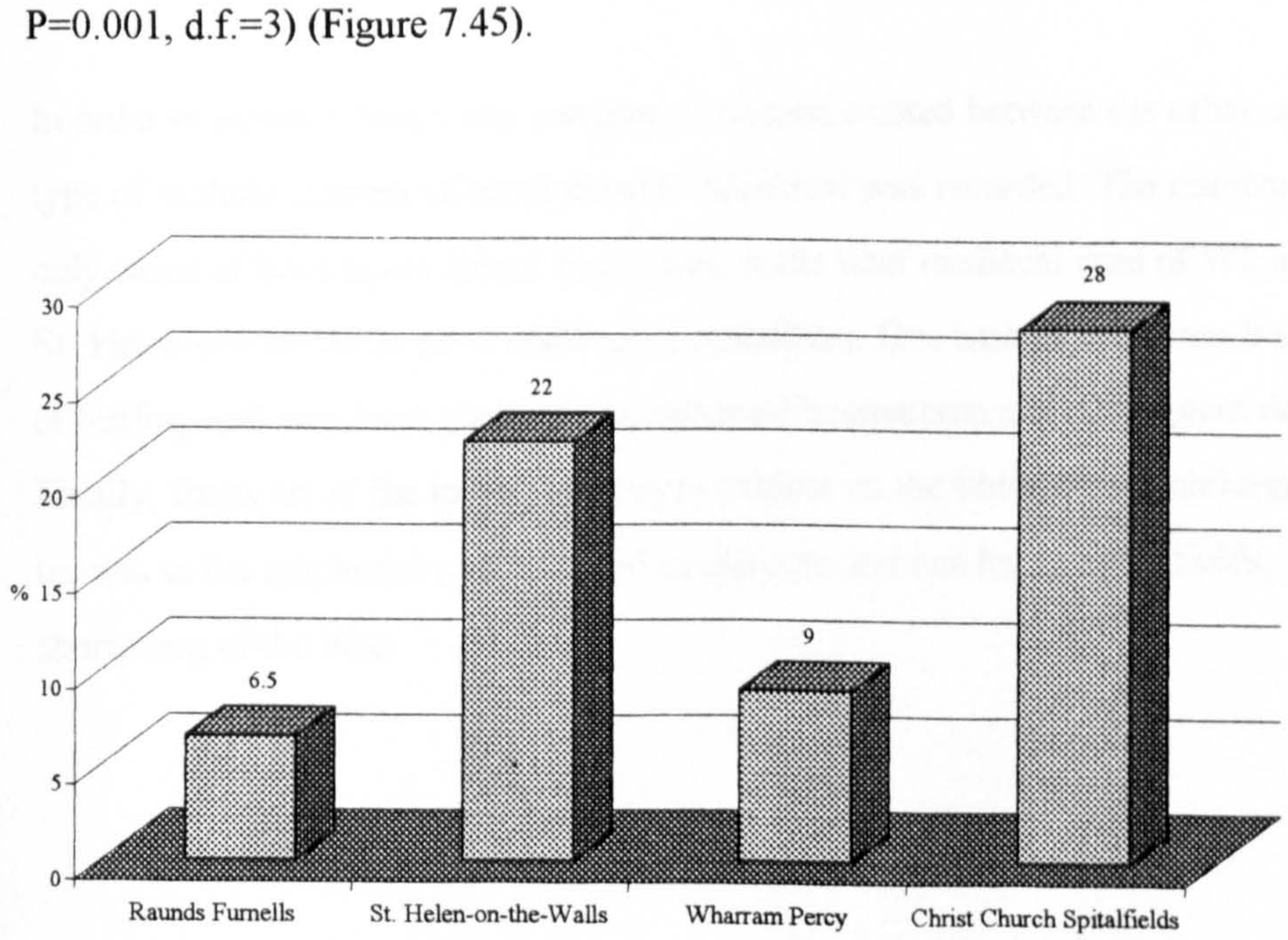

Figure 7.45 Prevalence of dental disease

7.7.3 TRAUMA

Figure 7.46 suggests that there were no obvious differences in the rates of trauma between the urban and rural sites, with industrial Spitalfields and agricultural Wharram Percy both having evidence for trauma in 33% of individuals.

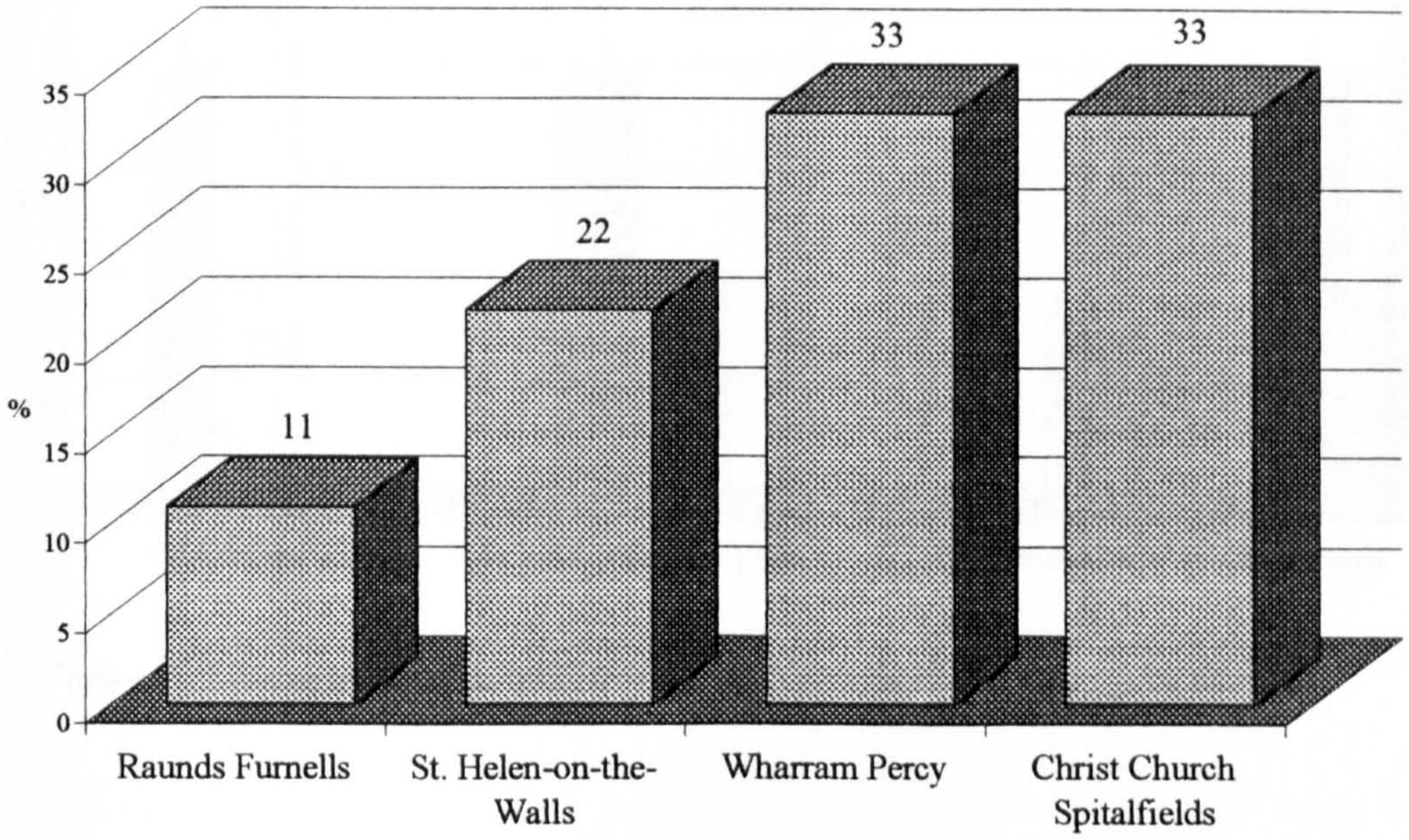

Figure 7.46 Trauma prevalence rates

In order to assess whether any patterns of trauma existed between the urban and rural sites, the type of skeletal element affected in each individual was recorded. The numbers are small but the only cases of head injury (blunt force) was in the later medieval sites of Wharram Percy (n=3) and St. Helen-on-the-Walls (n=1 cranium, 1 mandible). One case at Wharram Percy had no evidence of healing and may have resulted in a subdural haematoma and subsequent death of the child. Finally, fractures of the lower limbs were evident on the tibiae of two children from St. Helen, and trauma to the epiphyses was recorded in one arm and one leg at Spitalfields, resulting in shortening of the limb.

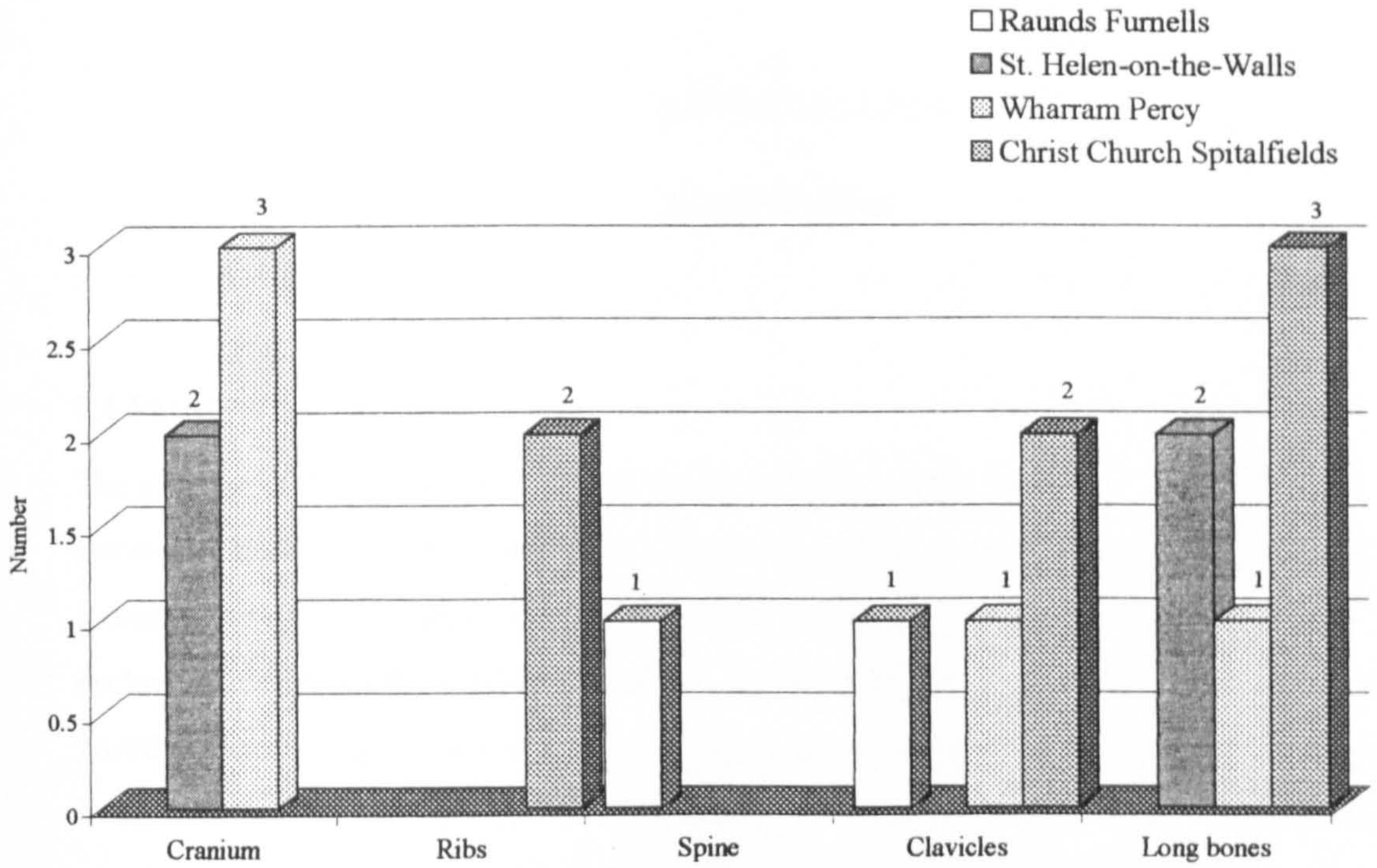

Figure 7.47 Number of individuals with trauma, by skeletal element.

CHAPTER EIGHT

DISCUSSION

8.1 INTRODUCTION

The purpose of this study was to assess the health of non-adults buried in urban and rural cemeteries between AD 850-1859. It was hypothesised that the development of urbanisation and industrialisation, with subsequent overcrowding and environmental pollution, would result in a decline in human health in the urban groups. This should be evident in lower mean ages at death, retarded growth, higher rates of childhood stress, and an increase in the morbidity of the children living in the urbanised environments. Any failure to find differences between the urban and rural populations may have several implications. Firstly, at certain stages in British history urbanisation was not as developed or as detrimental to human health, as the archaeological and documentary evidence would suggest. Secondly, it may indicate that the use of skeletal material to address these questions is not valid and that the commonly employed 'indicators of stress' are in fact, poor measures of environmental and cultural change.

This chapter examines the evidence for child health to assess whether any differences between the urban and rural environments was evident, and discusses these differences taking into account the historical, cultural and archaeological data. Finally, the ability of indicators of stress to measure environmental change will be examined in the light of the 'osteological paradox' (Wood *et al.*, 1992).

8.1.1 LIMITATION OF THE SAMPLES.

In 1988, Bogin suggested that any detrimental effect of the urban environment on human health could be measured by comparing the health of the urban population to their rural sedents. However, in archaeological samples it is not possible to distinguish the individuals who have migrated to an urban area from those who were born, raised and died in the urban environment. One of the reasons non-adults were chosen as the focus for this study was because it was initially believed that, in the past, children did not migrate and would die and be buried in the environment in which they were born. Therefore, any pathological indicators would be the direct result of living

in either an urban or a rural environment. However, children did migrate. For example, as infants of wealthy parents, the children from Christ Church Spitalfields may have been sent to the countryside to be wet-nursed (Fildes, 1988). If they survived, therefore, any evidence of early childhood stress may have been the result of the pathogens they encountered in their weaning environment (i.e. a rural one), and not in their final place of death and burial. Likewise, in the later medieval period, children from the age of seven were often sent from their villages into the towns and developing cities to find work as apprentices (Goldberg, 1986). Pelling (1988) has reported that these young migrants were particularly susceptible to disease in the urban environment because of a lack of exposure to specific pathogens, and many returned home to die. Therefore, although many may have contracted acute diseases such as smallpox, which of course would not be evident on the skeleton, after the Anglo-Saxon period, it cannot be assumed that all of the chronic infections recorded in rural graveyards were the result of encounters with the rural environment.

The cemetery samples used for this study not only differ in their environmental context, but the people living in them had different socio-economic statuses. As growth is more profoundly affected by poverty than environment (Tanner and Eveleth, 1976), this should be taken into account when comparing the skeletal growth profiles from different sites. However, it should also be remembered that the four samples represent populations from long periods in each of the site's histories. Therefore, assumptions about the socio-economic status of the children's parents come from particular periods of time. For instance, the parishioners of St. Helen-on-the-Walls were considered poor in 1420, but may have had better fortune in the preceding three hundred years of the cemetery's use. The children buried at Spitalfields, by virtue of their inclusion in the crypt, are considered to come from wealthy families, who may have shielded their offspring from any hazards facing the less affluent children living in industrial London. However, the merchants in this area were also known to suffer periods of financial hardship (Molleson and Cox, 1993).

All the samples showed some evidence of infant under-representation (see Chapter Five), making the analyses of mortality in this particularly sensitive portion of the sample difficult. Reduced sample sizes also have an effect on the interpretation of the growth profiles, particularly during later childhood, where fewer children die and enter the skeletal record. As cross-sectional data, neither 'growth' nor the 'velocity' of growth can ever be truly ascertained, and it can only be measured in many different children, at one particular time; the time of their death.

Finally, all of the children in this study are 'non-survivors.' They failed to adapt to the pressures of their environment and to reach adulthood. Therefore, any attempts to project the information gathered from these samples to infer the health of all the children living and growing-up in urban and rural environments in the past should be treated with caution.

8.2 URBAN-RURAL DIFFERENTIALS IN CHILD HEALTH

8.2.1 MORTALITY

8.2.1.1 The Infants

Despite the under-representation of the infants in the four samples, the pattern of mortality of the perinates from each site was expected to be different. Vögele (1994) suggested that in the urban environment, premature births were more common due to the pressures of work placed on the mothers. Therefore, deaths of children before the ages of 38-40 weeks of gestation (i.e. birth) might have been expected at St. Helen-on-the-Walls and Spitalfields. However, the assumed wealth of the Spitalfields mothers and medical advances, such as the introduction of the forceps in the early eighteenth century (Wear, 1992), may have resulted in greater numbers of children surviving birth. Once born, historical studies of infant deaths rates have shown that in some rural areas, the majority of deaths occurred due to 'endogenous' factors, resulting in high numbers of children dying at birth or within the first few days of life. In urban areas, however, post-neonatal deaths tended to outweigh neonatal deaths due to 'exogenous' causes arising from encounters with the external environment (Landers, 1990; Malhotra, 1990; Saunders, 1992; Vögele, 1994).

The infant death profiles of Raunds Furnells and Spitalfields resembled that of Gibson and McKeown's data (1951) (Figure 8.1a), suggesting the inclusion of stillbirths as well as infant deaths in the skeletal samples (Figure 7.1, p.120). At Wharram Percy, the age distribution was much flatter than expected for a normal mortality profile, with a peak at 35-37 weeks, indicating that a greater number of premature births were included at this site. If this were true, then the hard agricultural labour endured by the rural women may have resulted in this pattern. At St. Helen, however, the majority of the children were dying between 38 and 40 weeks, a pattern similar to that of Romano-British samples suspected of practising infanticide (Mays, 1993; Smith and Kahila, 1992) (Figure 8.1b).

Documentary evidence from St. Helen has suggested that many of the female tenants were involved in prostitution (Palliser, 1980). It is possible that the high number of neonatal deaths represent the disposal of unwanted births by their impoverished working mothers. However, they may also reflect the burial practises of the medieval period, where non-baptised infants were buried in a particular area of the cemetery. The rest of the infant burials may lie in the unexcavated portion of the site (see Chapter Five). However, detailed plans of the excavation are not available and, therefore, it is impossible to identify where in the cemetery the recovered infants were located.

Figure 8.1a Birmingham (1947)

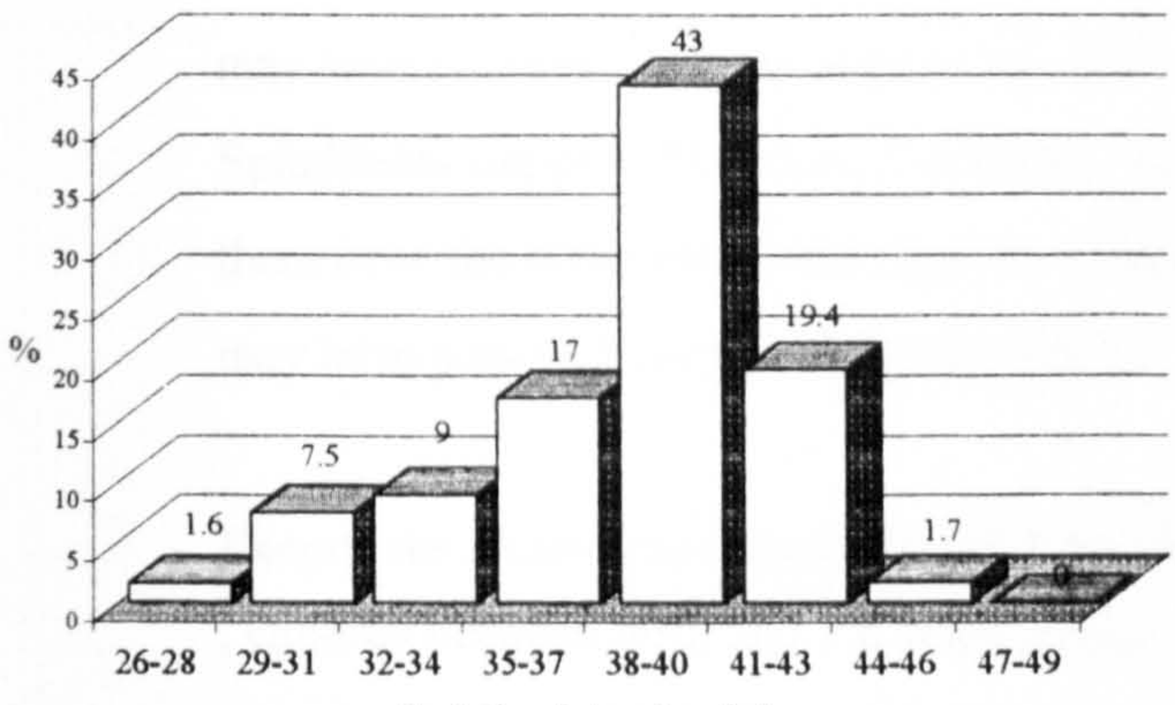

Figure 8.1b Romano-British Sites (Mays, 1993)

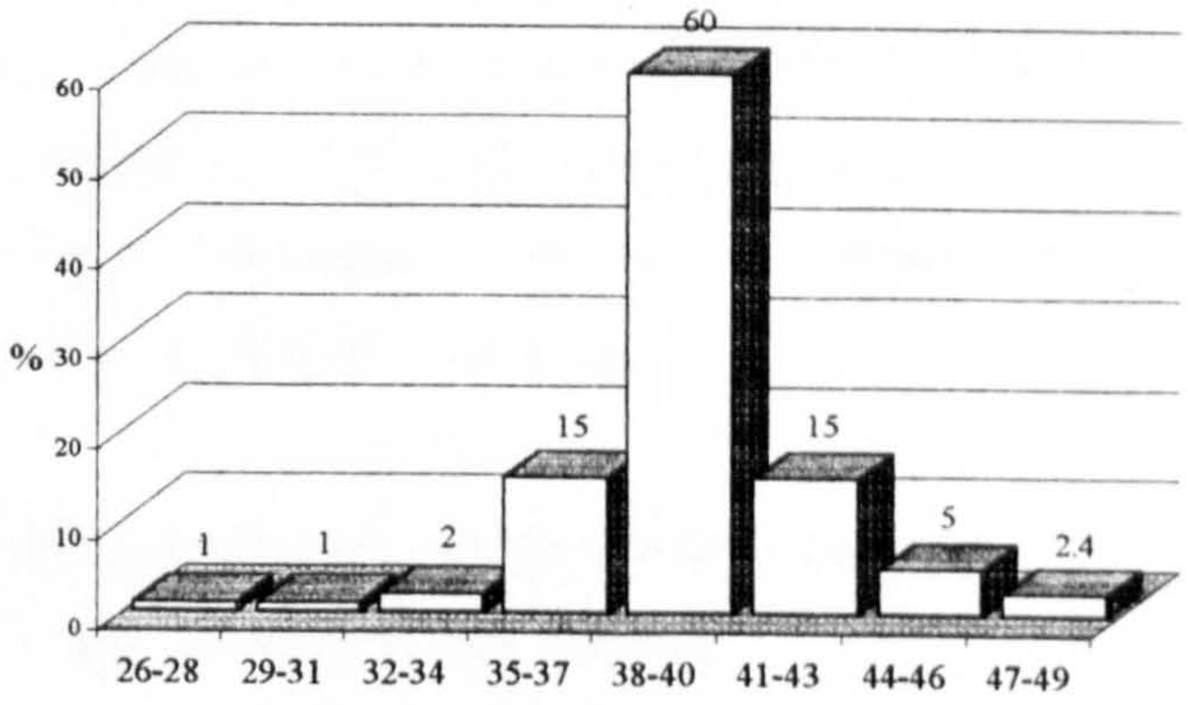

Figure 8.1a Percentage of stillbirths and deaths in the first year of life, in Birmingham in 1947 (n=920), from Gibson and McKeown (1951). Figure 8.1b Pattern of perinatal deaths in Romano-British sites (n=164) from Mays (1993).

However, the interpretation of these profiles must be treated with some scepticism. A peak at 38-40 weeks in perinatal mortality derived from archaeological samples, has recently been shown to be an artefact of the standards used to age the perinates (Gowland, 1998). The small sample sizes and regression equations employed by Scheuer and colleagues (1980) means that the archaeological samples are more likely to mimic the pattern of the standard used, which in this case leads to peaks during the 38-40 week period (Gowland, 1998). The small sample size at St. Helen-on-the-Walls (i.e. 12 perinates) may make this central skewing more pronounced, and so any interpretation of infanticide should be treated with caution.

When the proportions of neonatal and post-neonatal deaths were compared (Figure 7.2, p.122), the rural sites of Raunds Furnells and Wharram Percy had higher rates of neonatal mortality

compared to post-neonatal mortality, suggesting that endogenous causes of infant death were more common in rural areas during the medieval period, just as they were in the nineteenth century. and are today. St. Helen showed the greatest number of neonatal deaths (67%) but this is a reflection of the pattern discussed above. Only the figures from Spitalfields in London showed a higher prevalence of post-neonatal deaths (60%) than neonatal deaths (40%), indicating that the environmental factors associated with industrialisation had a greater impact on infant mortality than any health factors present at birth. This may also reflect successful birthing techniques that allowed children to survive the 38-40 week period.

8.2.1.2 The Children

It was expected that the environmental changes associated with urbanisation and industrialisation may have resulted in higher rates of mortality from acute and chronic diseases in the St. Helen and Spitalfields samples. However, it was also possible that the wealth of the latter group shielded them from the detrimental effects of their environment. Clean air and a ready access to fresh food may have provided favourable conditions for child survival in the rural areas.

Despite the under-representation of the infants, the high probability of death in the Spitalfields children between birth and 2.6 years of age is similar to that seen in Bradford in 1856 (Figure 7.5, p.128). In addition, the fall in infant deaths at Bradford between 1856 and 1931correlates with the documented decline in child mortality with social reforms in the early 1900's (Bogin, 1988a).

The pattern of mortality in the later medieval samples is more unexpected. Between the ages of 6.6 and 10.5 years the probability of death for the St. Helen and Wharram Percy children increases. Records from industrial Bradford indicate that, in 1856, only seven children died between the ages of six and ten years and only four died in 1831, suggesting that deaths in this age group are unusual, even in one of the most impoverished areas of industrial Britain. This pattern was identified by Grauer (1989) and Margerison (1997) in previous studies of the St. Helen sample, but has yet to be explained. A review of other skeletal samples from medieval York showed that at Jewbury, five to ten-year-olds made up 35% of the non-adult sample (Lilley *et al.*, 1994) and, at Fishergate, 24% of the non-adults were aged between 5 and 10 years (Stroud and Kemp, 1993). At St. Helen, 25% of the non-adults were aged between 6.6 and 10.5 years compared to 15% at Spitalfields and 13% at Raunds Furnells. At Wharram Percy the peak is

slightly earlier, with 28% of the children dying between the ages of 2.5 and 6.6 years. Therefore, the other later medieval sites at York are showing a similar increase in mortality between the ages of six and ten years.

In the later medieval period, many children from the age of seven were put to work as apprentices and travelled to urban areas to find work (Pelling, 1988). The health problems of urban-rural migrants have been discussed in Chapter Two, and exposure to an urban environment for teenagers migrating to York may explain the high probability of death at this age. The earlier peak in children aged between 2.6-6.5 years at Wharram Percy may reflect an earlier period when children were put to work on the farm. At both sites, the prevalence of periostitis, trauma and sinusitis peak during this age category and, at St. Helen, there was an increase in the prevalence of enamel hypoplasias. Therefore, an increase in their probability of death may be related to the hazards they encountered when they began employment.

8.2.1.3 Summary

An analysis of the mortality of the infants and children suggests that despite their relative wealth, the industrial environment contributed to more deaths in infancy and early childhood at Spitalfields than at the other sites. Although it appears that death during the birth period may have been less common, perhaps due to medical advances, the greater number of post-neonatal deaths at Spitalfields suggests that exposure to the industrial environment was more hazardous. Although it is tempting to suggest that mothers were practising infanticide at St. Helen, and that there were higher rates of premature births at Wharram Percy, this evidence is tentative. However, the unexpected peak in mortality of the children between six and ten years in the later medieval sites suggests that they were more susceptible to environmental hazards at this time. At St. Helen. the increase in the deaths of children with enamel defects during this period may suggest that they were already biologically damaged or of a weaker constitution (Goodman and Armelagos, 1988) and therefore could not rally against the stresses caused by their employment as apprentices.

8.2.2 GROWTH

Today, growth is commonly used as an index of overall community health, and as a guide to the population's ability to adapt to their environment. Studies of the differences in height between children in towns and villages have been carried out since the 1870 s and, before the 1900 s, it was consistently found that the height of rural children surpassed that of their urban peers

(Meredith, 1982). A reverse in this trend in the early twentieth century is often attributed to improvements in sanitation, nutrition and health care in the cities that surpassed those in rural areas. Today, in modern industrial countries differences between growth in urban and rural areas are barely discernible and poverty is believed to have a greater effect on growth, regardless of the environment (Tanner and Eveleth, 1976). It was expected that, due to new health hazards posed by developing industry, air pollution and overcrowding, the children from the urban areas would have been small compared to the children from the rural environment. In addition, as a result of their apparent low socio-economic status, the children from St. Helen would also be shorter than those from Spitalfields.

The similarity between the growth profiles of the later medieval populations is intriguing and appears to suggest that, either the urban environment had no detrimental effect on growth or, that during the medieval period environmental factors were similar in the urban and rural communities. Some researchers have taken the numerous official edicts on public health in medieval England as evidence that medieval cities were not actually filthy. They argue that the inhabitants were aware of the health hazards caused by the build-up of refuse, and that images of medieval squalor were the creation of Victorian propaganda, designed to take attention away from the appalling conditions of the industrial cities (Lord, 1997; Thorndike, 1928). However, it may be that at St. Helen, wage-earning mothers were able to provide their children with adequate nutrition for growth. Later in their lives these urban children may have become apprentices in the industries of the neighbouring parish of Bedern, and would have been provided for by their employers (Pelling, 1988). In addition, Bogin (1988a: 156) has argued that: '*urban children, whose parents are engaged in wage-labor, work less at energy intensive labor than the rural children, whose parents are engaged in agricultural labor.*' Therefore, the lower than expected growth profiles of the Wharram Percy and Raunds Furnells children may be the result of the hard manual labour associated with the agricultural environment.

The differences between the St. Helen and Spitalfields children suggest that despite their cultural advantages in wealth, the children living in industrial London were smaller at birth, and after puberty, than the children from urban York. This indicates that it was the rise of industrialisation, and not urbanisation, that took its toll on human health.

8.2.2.1 Growth at the Onset of Puberty

From the age of 12 years, the growth profiles of the sites begin to deviate (Figure 7.12, p.136) with the children from the later medieval period surpassing the growth of the other samples (Figure 8.2). By 13 years of age, the St. Helen adolescents achieved a higher percentage of their final femoral length before their death (94%) than any of the other children. At Wharram Percy, the increase was more gradual, but the children attained 93% of their expected femoral length by 16 years of age. Although the profiles consisted of estimated femoral lengths between 13 and 14 years of age, neither the Spitalfields nor the Raunds Furnells children show an increase in growth at 12 years. Despite an apparent spurt in growth between 11 and 12 years of age in the Spitalfields children (from 74% to 84% of final expected bone length), by 15 years they, and the children from Raunds Furnells, are strikingly smaller than the later medieval children by the time of their death.

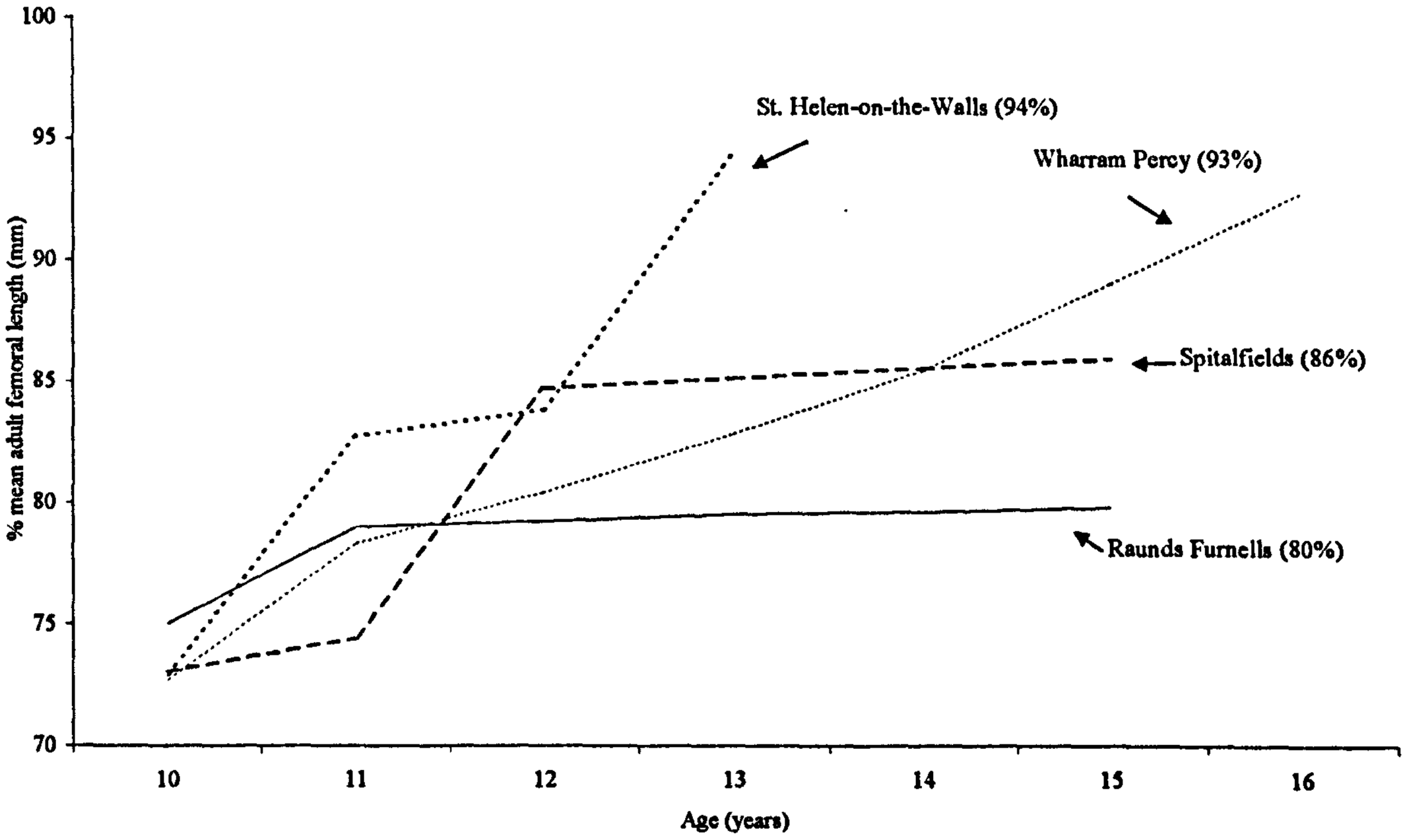

Figure 8.2 Percentage of attained adult femoral length (from 10 years).

Studies of growth profiles of non-adult skeletons in the later age groups are always problematic due to smaller sample sizes, and the inability to assign a sex to the skeletons at a time when sexual dimorphism plays an important role in growth. However, this growth period is crucial in assessing morbidity because any growth retardation present at the onset of puberty cannot be 'caught-up.'

Today, females begin puberty, and thus their adolescent growth spurt, nearly two years earlier than males, who finally surpass the girls in body size (Eveleth and Tanner, 1990). Several medieval sources indicate that the age of menarche for females in the late medieval period was between 12 and 14 years and that boys matured a few years later, between 14 and 16 years of age (Danker-Hopfe, 1986). It is possible that the apparent difference in the timing of the growth spurt between the sites was the result of different proportions of males and females. However, with the exception of Wharram Percy, the samples showed a preponderance of females in the 17-25 year age bracket (Boddington, 1996; Dawes and Magilton, 1980; Mays *pers. comm.*; Molleson and Cox, 1993) (Table 8.1).

Table 8.1 Percentage of males and females in the 17-25 year age category.

	Raunds Furnells	*St. Helen*	*Wharram Percy*	*Spitalfields*
Male	39	42	55	40
Female	61	56	45	60

The results suggest that at both Spitalfields and Raunds Furnells the children display a later growth spurt and retardation in growth. Such delays are often due to mal- or undernutrition that may result in a later but longer period of growth, and a delay in skeletal maturation (Eveleth and Tanner, 1990). Other studies that have incorporated Raunds Furnells into their growth data have shown these Anglo-Saxon children to be among the smaller in their studies, particularly in the later age groups (Hoppa, 1992; Saunders *et al.*, 1993). However, as we know nothing of the social-status of the individuals interred at Raunds Furnells it is difficult to understand why they should have different growth profiles from children at rural Wharram Percy.

8.2.2.2 Summary

The results suggest that there were no differences in the growth of the children from the later medieval period, that these children shared a similar adolescent growth spurt at 12 years of age, and managed to attain up to 94% of their expected adult size before their death. However, although urbanisation does not appear to have been affecting the growth of the St. Helen children, industrialisation did have a profound effect on the growth of the children from Spitalfields, who were smaller and appear to have suffered a delay in puberty. Similarly, the children from Raunds Furnells, although not significantly different to the rural children from the later medieval period, were smaller and suffered a similar delay in their adolescent growth spurt. Therefore, this study

suggests that it was industrialisation, and not urbanisation in the later medieval period, that had a detrimental effect on the growth of the children.

8.2.3 MORBIDITY

It was expected that any detrimental effect of the urban environment would result in a higher prevalence of stress indicators in the urban children. However, chi-squared tests revealed that there were no significant differences between the sites, although the children at St. Helens had the highest prevalence of periostitis, enamel defects and sinusitis. These results suggest that these popular markers are, in fact, poor measures of environmental stress.

By examining the prevalence of these indicators in more detail, however, the difference between the urban and rural samples becomes more apparent. Both urban sites had evidence of enamel hypoplasias on the deciduous dentition, indicating stress *in utero* as a result of poor maternal health. In the eighteenth and nineteenth centuries it was common for wealthy women to have large numbers of children with short birth intervals. The use of wet-nurses and high infant mortality meant that women were frequently pregnant, resulting in nutritional deficiencies in the mother, and stress for the developing child. However, the presence of deciduous tooth hypoplasias also suggests that urban children were capable of surviving a stressful *in utero* environment for at least one year after their birth.

8.2.3.1 The Weanling's Dilemma?

In the seventeenth and eighteenth centuries, mean weaning ages in Britain were documented to have fallen from 18 to 7 months (Fildes, 1996). Therefore, it was expected that at Spitalfields, lower weaning ages would be evident in an earlier frequency of cribra orbitalia, enamel hypoplasia formation and early growth faltering. In addition, the increased potential of food contamination and infection in the urban environment was expected to be evident in the infants from St. Helen-on-the-Walls. The results showed that, at Spitalfields, the majority of the enamel hypoplasias were formed around six months of age, and that developmental ages increased in half year intervals from the urban to the rural sites, with the earliest site of Raunds Furnells showing the latest peak at two years (see Table 7.11, p.156). The peak in cribra orbitalia in Spitalfields was also earlier than at the other sites, occurring between the ages of six months and two years, and corresponds to the high probability of death during this period. These results suggest that the children at

Spitalfields were being weaned at around six months of age, as suggested by the documentary evidence.

King and Ulijaszek (1999) have argued that, after six months, the nutritional and immunological benefits of breast milk begin to decline. Therefore, if the child's diet is not successfully supplemented by nutritious and uncontaminated food, then any subsequent undernutrition and infection should be evident in growth faltering in the infants and young children. At Spitalfields, the young children are consistently smaller than those from the other sites but there was no evidence of growth faltering after the six-month period, when the stress indicators suggest that weaning began (Figure 8.3).

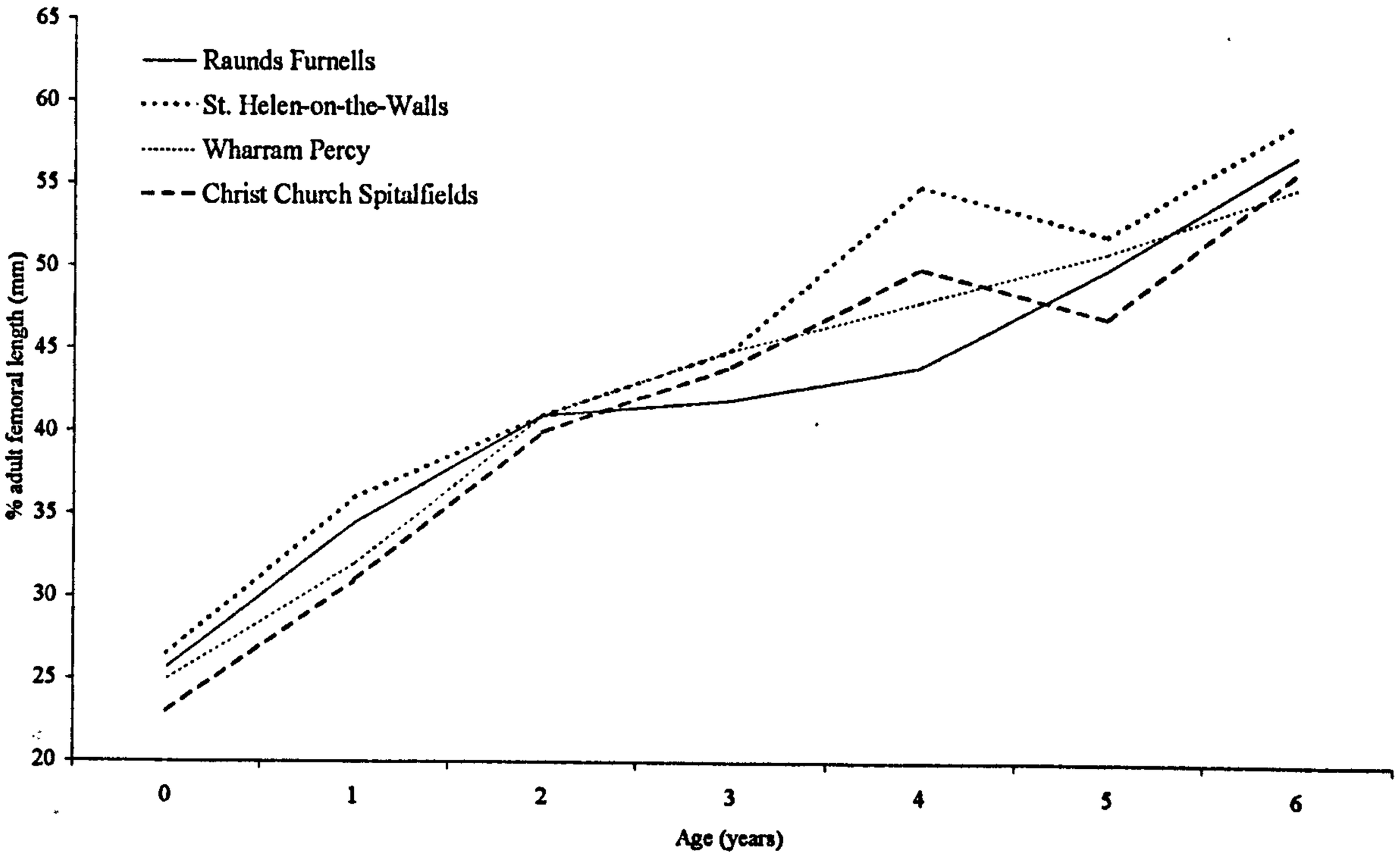

Figure 8.3 Skeletal growth profiles of children between birth and six years of age.

In both the later medieval samples there was an increase in enamel hypoplasia formation after one year of age suggesting that, although supplementation must have been initially successful, the children later succumbed to environmental pathogens. However, the growth patterns of the St. Helen and Wharram Percy children do not fluctuate at one year of age, or at 2.6 years, when the prevalence of cribra orbitalia increases. The peak in long bone length at Spitalfields and St. Helens at four years is an artefact of the small samples sizes in these groups rather than evidence of accelerated growth (see Table 7.4, p.131). However, at Raunds Furnells, growth between two and four years of age does falter, corresponding to a peak in the formation of enamel hypoplasias.

It is possible that this apparent 'faltering' is the result of the smaller number of individuals contributing to the growth curves at four years, but there is other evidence to support a later weaning age in this Anglo-Saxon site.

At Raunds Furnells, healed cases of cribra orbitalia, suggesting a recovery from iron deficiency anaemia, did not occur until the 2.6-6.5 year age category, compared to 0.6-2.5 years at the other sites. This recovery is also marked by an increase in expectation of life between 2.6 and 6.5 years (Figure 7.6, p.129). During this age category active new bone formation also becomes more prevalent and, again, active periostitis occurs earlier in the other groups. The tentative evidence of growth retardation at the age of two years, coupled with active infections, iron deficiency anaemia and the occurrence of enamel hypoplasias points to weaning stress. If it is occurring later in the Anglo-Saxon population, then it appears that a delay in introducing new food to the child's diet resulted in more detrimental effects to their health than earlier weaning did in the other groups. At the other sites weaning stress, suggested by the dental defects and evidence for anaemia, are not substantiated by growth faltering and this may imply that food supplementation was relatively successful. It is possible that the malnutrition and infection did affect growth between the ages of one and two years but that this evidence was obscured by catch-up growth of the children entering the later age category. However, it seems unlikely that a child would experience accelerated growth just before their death, unless they died unexpectedly as a result of an accident or an acute infection.

8.2.3.2 Cribra Orbitalia

Up to 60% of the children in all the samples had evidence for cribra orbitalia. As expected there was a greater prevalence of active lesions than healed lesions in the non-adult skeletons, suggesting that in most cases, whatever caused the lesion persisted until their death. Cribra orbitalia is considered to be indicative of iron deficiency anaemia as a result of either malnutrition or a high pathogen load (Stuart-Macadam and Kent, 1992). The association of this lesion with new bone formation, supports the theory that the children suffered anaemia due to infection, and agrees with the findings of Mensforth and co-workers (1978) and Palkovich (1987). The high percentage of cribra orbitalia is also consistent with other studies: at later medieval Fishergate 64% of the non-adults had the lesions (Stroud and Kemp, 1993), and in later medieval Spain De la Rúa and colleagues (1995) found evidence of the lesions in 55.5% of the children. Although there was no difference between the prevalence of cribra orbitalia at the sites, the majority of the lesions

at Wharram Percy were considered to be mild (grade 2) and the more severe form of the lesions were found in the urban samples.

8.2.3.3 Metabolic Disease

The most striking difference in morbidity between the sites was the high prevalence of metabolic disease in the children from industrial London (54%). By the time it was first described in 1650, rickets had become a disease of urbanisation and was so prominent in the industrial and mining areas of Britain it was known as the 'English Disease' (Mankin, 1974). Initially, rickets and scurvy were diseases of the rich who could afford to wean their children on fashionable formulas and condensed milk which were low in vitamins C and D (Fildes, 1986a). In addition, high levels of air pollution in the industrial environment would have restricted their exposure to sunlight resulting in rickets. Despite the high prevalence of rickets and scurvy at Spitalfields, endocranial lesions were less common; this indicates that endocranial haemorrhage as a result of scurvy was not a significant aetiological factor in the formation of these lesions. However, as metabolic disease was shown to have a significant effect on the growth of affected individuals (Figure 7.44, p.164) this indicator goes some way to explain the short stature of the Spitalfields children.

The paucity of evidence for metabolic diseases at Raunds Furnells (6%) and, more surprisingly, at urban St. Helen (6%), is probably due to the poor preservation of the necessary skeletal elements (ribs and skull) rather than any dietary and environmental advantages at these sites. However, evidence for rickets at Wharram Percy (14%) was unexpected, and Ortner and Mays (1997) have suggested that these children may have been sickly, kept indoors and hence were deprived of sunlight which resulted in rickets occurring shortly before their death.

8.2.3.4 Infection

Although the presence of rickets and scurvy in the younger individuals may have disguised any non-specific new bone formation, the lack of periostitis in the older individuals at Spitalfields suggests that these children were not exposed to trauma or infection later in their life. This may be due to their social status which protected them from exposure to chronic diseases; however, it may also indicate that these children were not strong enough to sustain a chronic infection, and died in the acute stages of a disease before any bone lesions could occur.

8.2.3.5 Dental Disease

Several studies in Britain have shown an increase in dental disease with time (Moore and Corbett, 1973; 1975; 1976). The results suggest that the urban children had access to a more cariogenic diet, perhaps reflecting the accessibility of imported and refined foods, and they had lower levels of dental hygiene, despite the difference in their social status. Dental disease was also associated with enamel hypoplasias and maxillary sinusitis. Defective enamel on the occlusal surface of the molars would leave a tooth susceptible to invasion by cariogenic pathogens which, in turn, could lead to a spread of infection into the neighbouring maxillary sinuses.

8.2.3.6 Respiratory Diseases

Complaints about poor air quality in the medieval period, the burning of coal, and air pollution with the rise of industrialisation, was expected to be evident in a high prevalence of respiratory diseases in the children living in the urban and industrial environments. However, despite previously finding a higher prevalence of maxillary sinusitis in the adults from St. Helen, compared to those at Wharram Percy and Raunds Furnells (Roberts and Lewis, 1994, 1995; Lewis, *et al.*, 1995), no significant differences were evident in the non-adults. It is possible that the difficulty in diagnosing the lesion in growing sinuses, and the problems of viewing intact sinuses, obscured any differences should they exist. However, the low prevalence of maxillary sinusitis at Spitalfields (3% of sinuses examined) is particularly surprising considering that the industrial period was notorious for environmental pollution. Nevertheless, a low prevalence of respiratory disease was also found in the adults in this sample (Roberts, 1998). In fact, once the evidence for dentally induced sinusitis had been accounted for, Wharram Percy had the highest rate of sinusitis resulting from other factors. Other evidence for respiratory infections such as rhinitis and pulmonary infections was also higher in Wharram Percy and Raunds Furnells. Although this result was initially surprising, Williams and Galley (1995) found respiratory diseases to be twice as common in rural than urban areas. Allergic reactions brought about by close contact with animals and exposure to irritants in the soil may be responsible.

Documentary evidence for unhygienic urban cowsheds, harbouring tuberculosis, meant that high levels of gastrointestinal TB might have been expected at St. Helen and Spitalfields. However, despite all of the sites having some evidence of tuberculosis, only one case (Spitalfields 2691) appeared to be pulmonary in origin (See Appendix X). This suggests that all the children were exposed to the harmful effects of infected milk and is in agreement with the medical and historical

evidence (Griffith, 1919; Cronjé, 1984; Atkins, 1992). Of course, there may have been many more cases of the pulmonary disease that never became visible on the skeleton, or may have only resulted in rib periostitis before the child succumbed to the disease and died.

8.2.3.7 Trauma

A recent study has suggested that rural areas are more physically hazardous environments, with Judd and Roberts (1999) reporting higher rates of trauma in adults from Raunds Furnells compared to those at St. Helen-on-the-Walls, and Blackfriars, in medieval Gloucester. Accidental deaths as a result of drowning and suffocating may not be identified in skeletal material, but difference in the chores of urban and rural children may be evident in the bones affected and the types of injury received.

There was little evidence of trauma in the non-adults and it is possible that many of the injuries they may have received were greenstick fractures thatusuallyheal quickly without trace of deformity. Although there was no difference in the number of cases of trauma between the sites, only the later medieval sites had evidence for head injuries, which may indicate occupationally related trauma, or even child abuse. One of the cases at Wharram Percy had no evidence of healing, and probably resulted in a subdural haematoma and subsequent death of the child. Interestingly, only the Spitalfields children had evidence of rib fractures, associated with a direct blow to the chest, either from a fall or due to interpersonal violence (Roberts and Manchester, 1996). At Spitalfields, two children had fractured ribs and in one case they were in the process of healing. Despite their higher social rank, this may provide tentative evidence for child abuse in the industrial group, and warrants further investigation. Another interesting feature of the Spitalfields sample was the evidence for surgical procedures in three of the children, which provides evidence for a growing interest in the causes of death of children that would culminate in the establishment of children's hospitals in 1850 (Lomax, 1994).

8.2.3.8 Summary

Although there were no significant differences in the prevalence of indicators of stress between the sites, when the severity and age profiles of the lesions were compared differences did emerge. Deciduous tooth hypoplasias pointed to maternal stress in the urban populations, and higher rates of dental disease were apparent, suggesting access to refined food and low levels of dental hygiene in the urbanised areas. Spitalfields appears to have had the lowest weaning ages, at around one

year of age, corresponding to the documentary evidence at the time, followed by St. Helen. Wharram Percy and finally Raunds Furnells. Despite the evidence for enamel hypoplasia formation and cribra orbitalia, only the growth curves at Raunds Furnells showed evidence of growth faltering at a time when the children were suspected of being weaned. This disagreement between the stress indicators and growth profiles is disconcerting. The presence of stress indicators suggests that weaning was not entirely successful, and that the children did experience some malnutrition and infection; however , this is not substantiated by the growth profiles. Perhaps higher levels of stress are needed to influence growth than are needed to cause dental defects and anaemia.

Rickets and scurvy were rife in the Spitalfields children, resulting in their smaller growth profiles compared to their peers from the other sites. These high levels of metabolic disease, indicative of deficiencies in vitamins C and D, were perhaps due to the use of condensed milk for infant feeding and lack of sunlight as a result of air pollution. However, it was the rural sites that showed the greatest evidence for respiratory infections, suggesting that the agricultural environment exposed these children to allergies and harmful pathogens in the soil. Chronic tuberculosis occurred with equal frequency in all the sites. Although there was very little evidence for trauma in the non-adults, the high occurrence of head injuries in the later medieval sites, particularly during the 6-10 years period, adds weight to the argument that they were beginning employment at this age. In addition, the tentative evidence for inter-personal violence in the Spitalfields sample deserves further investigation.

Finally, the evidence for the morbidity and mortality of these samples has shown that the divide between the urban and rural environments is not straightforward. The development of industrialisation resulted in high levels of metabolic disease, higher levels of infant mortality and growth retardation. However, in the later medieval period differences between the urban and rural environments were barely discernible, and environmental pollution in the towns during this period may not have been as damaging as the documentary evidence suggests. However, respiratory diseases were a particular problem in the agricultural communities. It is evident that both environments presented hazards for the children growing up in them.

8.3 THE 'OSTEOLOGICAL PARADOX' REVISITED.

Is it logical to assume that a group of skeletons displaying pathological lesions and other stress indicators represent the disadvantaged of the society from which they were derived? Wood and colleagues (1992) have argued that this basic assumption, commonly used to measure past population morbidity, is paradoxical. In order to display a stress indicator such as an enamel hypoplasia or a Harris line it is necessary to recover from the stress that resulted in a cessation of growth, as these lesions only become visible when normal growth has been resumed. Thus, it follows that only those with a strong immune system, and perhaps culturally advantaged, can survive the stress episode. In fact, shouldn't the 'weaker' or culturally disadvantaged individuals enter the mortality sample with no, or fewer, skeletal markers of stress and disease because of their inability to recover from an episode of stress?

For instance, the urban sites in this current study had evidence of enamel hypoplasias on the deciduous dentition indicating stress *in utero,* probably as a result of poor maternal health. It is interesting that the analysis of perinatal mortality showed that the children at Spitalfields were dying in the later weeks after birth, suggesting exogenous causes of death, and yet there is clear evidence for *in utero* stress that might be expected to affect the integrity of the newborn. Nevertheless, these children were able to survive long enough after birth to present with deciduous enamel hypoplasias, indicating that rather than being 'weaker' they were actually being born into conditions more favourable to their survival and were actually the 'stronger' individuals.

In order to assess those most at risk from stress it is important to be aware of the environmental and cultural factors that may buffer or expose the population to physiological disruption. Goodman (1993) argues that evidence from developing countries refutes the idea of the healthiest individuals having more stress indicators and states: '*I know of no situation in which a clearly advantaged group, living or past, has more hypoplasias than a disadvantaged group*' (1993: *284*). He suggests that by taking the mean age at death, the weakest individuals would be identified as those who died at an earlier age. Although stress indicators are generally unrelated to the final cause of death, the process causing the lesion may have affected the individual's immune system, leaving them more susceptible to a fatal infection. Therefore, individuals with stress indicators may have been selected to die at an earlier age, something that Saunders and Hoppa (1993) termed '*selective mortality.* ' Individuals with stress indicators should be entering the mortality record earlier than those who do not display the lesions.

In order to test these arguments, the impact of stress on the mean age at death and on the growth of individuals dying with and without stress indicators were examined. At all the sites, non-adults with enamel hypoplasias lived slightly longer than those without, probably reflecting the need for children to survive an episode of stress before a defect becomes visible (Table 7.8, p.147). At Spitalfields, individuals with new bone formation lived, on average, five years longer than those without evidence of infection and, at Wharram Percy and Raunds Furnells, individuals with cribra orbitalia lived one year longer than those without evidence of the lesions. Although these results are not consistent at each site, neither periostitis nor cribra orbitalia requires the individual to recover from stress for the lesions to be visible. Therefore, these results support the theory that, in order to display a skeletal marker, individuals must be disadvantaged enough to be exposed to and suffer from a condition, and yet strong enough to survive an infection into its chronic stages.

That stress indicators do not seem to have a consistent effect on growth is supported by previous studies (Mays, 1985; Ribot, 1992; Ribot and Roberts, 1996). This may be explained by the fact that lesions such as enamel hypoplasias and Harris lines only become visible on the teeth and bones when normal growth is resumed. If this is followed by 'catch-up' growth, then any differences that may have existed between the 'stressed' and 'non-stressed' individuals will be lost. The indicators of stress associated with on-going problems, such as infection or anaemia (new bone formation/ cribra orbitalia), were shown to have an effect on growth (Table 7.7, p.143). Where differences did exist, the pattern is for the 'stressed' individuals to be initially smaller than the 'non-stressed' individuals but to overtake them in height in the later age categories. Although the sample sizes for this analysis were small, the results suggest that although the children may have been initially small and therefore 'selected' to die, the circumstances that helped the individuals survive the stress episode, also helped them recover their growth trajectory and become taller.

8.3.2 SUMMARY

Evidence from the non-adults in this studyseemsto support the theory that in order to suffer stress chronic enough to be visible on the skeleton, an individual needs to have an immune system capable of surviving the acute stages of infection. However, they must also be culturally or biologically disadvantaged enough to be exposed to infection in the first place. Evidence from the growth profiles suggests that once an individual has recovered from the initial growth retardation

associated with childhood stress, they not only recover but exceed the growth of their non-stressed peers in later years. However, all of the children in this study failed to survive their environments into adulthood and, although they provide a detailed insight into survival during the period when childhood stress occurred, further research into the age at death of the adult individuals with stress indicators is needed to fully answer these questions.

CHAPTER NINE

CONCLUSIONS

9.1 THE IMPACT OF URBANISATION AND INDUSTRIALISATION

The primary conclusions of this research are that:

- Differences in the morbidity and mortality of non-adults from urban and rural environments did exist in the past
- It was industrialisation, rather than urbanisation, that had the greatest impact on child health
- Although they do not correspond well with growth profiles, skeletal and dental indicators of stress are effective measures of environmental change, when considered in relation to age at death and the age of their peak development.
- Hard agricultural labour, urban employment and changes in weaning ages and infant feeding practices contributed to differences in health in the urban and rural environments.
- Social economic status did not buffer children from the detrimental effects of the industrial environment

This study has illustrated the validity of using skeletal material to answer questions about population adaptation to changing environments. In particular, the non-adult skeletal remains can provide insights on growth and the impact of stress on survival in childhood.

Documentary and archaeological evidence suggests that the development of urbanisation in the later medieval period was accompanied by numerous complaints about environmental pollution

from the urban inhabitants. However, this study has shown that it was the industrialisation of the eighteenth century, rather than urbanisation, that had the greatest impact on child health and survival. Despite the relative wealth of their families, the industrial environment contributed to more deaths in infancy and early childhood at Christ Church Spitalfields than at later medieval York or the rural medieval sites. Growth retardation, exacerbated by the high prevalence of rickets and scurvy, also reflected the poor health status of the children living in industrial London. The age of occurrence of enamel hypoplasias, and peaks in cribra orbitalia, suggest that as societies became more industrialised weaning ages declined, with the Anglo-Saxon children being weaned

at two years compared to one year at Spitalfields. Deciduous hypoplasias pointed to maternal stress in the urban populations, and higher rates of dental disease were apparent, suggesting access to refined food and low levels of dental hygiene in the urbanised areas. However, it was the rural sites that showed the greatest evidence for respiratory infections, indicating that the agricultural environment exposed these children to allergies and harmful pathogens in the soil.

In the older children, growth profiles revealed a delay in the pubescent growth spurt at Spitalfields and rural Raunds Furnells when compared to the children from later medieval Yorkshire, indicating growth retardation at a late stage in their development. The common perception that children from the later medieval urban environment, with its overcrowded and poor sanitary conditions, would have suffered from malnutrition and disease has not been born out by the growth curves from the York sample. The similarity between the growth profiles of the later medieval populations is intriguing and appears to suggest that the urban environment had no detrimental effect on growth or, that during the medieval period, environmental factors were similar in the urban and rural communities. Migration and trade between these contemporaneous Yorkshire sites may be the reason for their similarity or, perhaps, the economic aspects of the urban centres that allowed women and children to become wage earners, was favourable. In contrast, hard manual labour and seasonal food shortages may have impaired the growth of the rural children. However, an increase in mortality, a peak in the prevalence of periostitis and sinusitis, and the occurrence of head injuries during the six to ten year age group at Wharram Percy and St. Helen suggests that they began employment, perhaps as apprentices.

Issues concerning the benefit of social-economic status have also been raised. Although the wealth of their parents may have been instrumental in protecting the Spitalfields children at birth it also allowed their parents access to fashionable formulas and may have limited their exposure to sunlight, resulting in rickets and scurvy. Although initially considered to be poor in comparison, the children from St. Helen had the best growth profiles and may have been buffered by their ability to earn wages as apprentices. Finally, it is evident that studies between urban and rural environments are not straightforward and different social classes, cultural practices, climate, genetics and the migration of peoples, make such studies both stimulating and challenging.

9.2 Recommendations for Future Research

Although previously neglected, there is great potential for the study of non-adult skeletons from archaeological sites. As more archaeologists are trained to recognise and recover these small remains further insights into the adaptation of a population to environmental change will be possible. Improved ancient DNA techniques may help us assign a sex to non-adults and allow us to address gender issues and develop questions about differential treatment of male and female children, particularly concerning infanticide, wet-nursing, education, disease susceptibility and age at death. Stable isotope analysis, analysing strontium/calcium ratios and nitrogen values in bone to pinpoint weaning ages has been carried out in North America but, as yet, has not been investigated in British populations. This type of research may help to substantiate the results of this study, which suggest that weaning times were reduced with industrialisation.

Although there are many conditions that can be identified on the non-adult skeleton, we need to analyse the timing and pattern of growth in children to help distinguish pathological lesions from normal new bone formation during growth and, to finally identify neonatal and infant diseases from both long bone and skull lesions. Fracture patterns, although limited by remodelling, may help identify child abuse or accidents and perhaps suggest the type of labour children were engaged in. The contrasting patterns of trauma in the four sites under study warrants further investigation. Although much work has been done on children's hospitals and their treatment since the 1920 s, prehistoric evidence has been neglected. Studies into the social history of medicine have tended to concentrate on the history of childhood, rather than their physical health. We need to know more about ancient medical knowledge concerning childhood disease and the treatment of children in the past to help interpret patterns of disease, particularly during the Anglo-Saxon period.

Finally, we need to standardise our ageing techniques and age categories for non-adult skeletal remains to enable comparisons from different sites to be carried out. Once the problems of preservation, methods of analysis and diagnosis have been resolved, broader questions about the treatment and health of children from different time periods and societies can begin to be answered.

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APPENDICES

Appendix I: Developmental Stages for the Deciduous and Permanent Dentition

Table 1. Developmental stages for the deciduous dentition.
Mean ages (years) averaged for males and females (after Moorrees *et al.*, 1964a)

Developmental Stage	*dc*	*dm1*	*dm2*
Cco	0.0	0.0	0.0
Coc	0.0	0.0	0.0
Cr½	0.3	0.0	0.3
Cr¾	0.5	0.3	0.5
Crc	0.8	0.4	0.8
Ri	0.8	0.5	1.0
Cli	-	0.6	1.4
R¼	1.0	0.8	1.5
R½	1.3	0.9	1.6
R¾	1.8	1.2	1.9
Rc	2.0	1.2	2.0
A½	2.5	1.5	2.5
Ac	3.0	1.8	2.9

Table 2. Developmental stages for the permanent dentition.
Mean ages (years) averaged for males and females (after Smith, 1991)

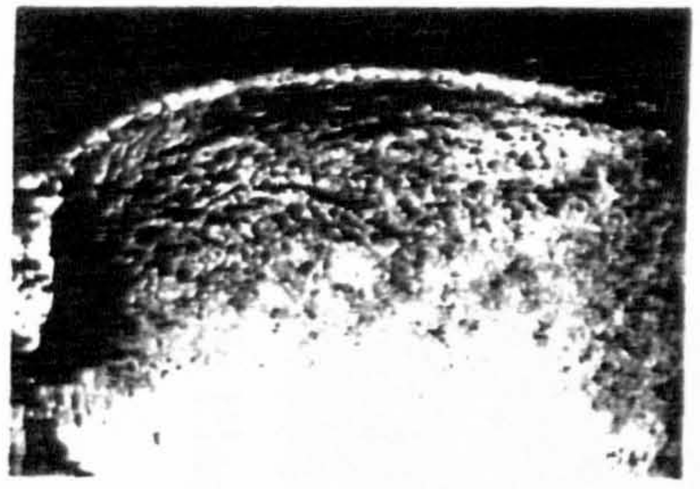
Developmental Stage	*I1*	*I2*	*C*	*P1*	*P2*	*M1*	*M2*	*M3*
Ci	-	-	0.5	1.8	3.0	0.0	3.6	9.4
Cco	-	-	0.7	2.3	3.5	0.2	3.8	9.9
Coc	-	-	1.3	2.9	4.2	0.6	4.4	10.5
Cr½	-	-	2.0	3.6	4.7	1.0	4.9	11.1
Cr¾	-	-	2.9	4.4	5.4	1.5	5.5	11.6
Crc	-	-	4.0	5.1	6.2	2.2	6.3	12.1
Ri	-	-	4.7	5.8	6.8	2.7	7.0	12.8
Rcl	-	-	-	-	-	3.5	7.8	13.6
R¼	4.5	5.0	5.5	6.7	7.6	4.5	9.3	14.6
R½	5.2	5.7	7.5	8.4	9.1	5.1	9.9	15.4
R⅔	5.7	6.4	-	-	-	-	-	-
R¾	6.5	6.9	8.9	9.5	10.4	5.8	10.9	16.4
Rc	6.9	7.8	9.5	10.2	11.1	6.1	11.4	16.9
A½	7.3	8.3	10.8	11.5	12.3	7.3	12.7	18.2
Ac	7.9	8.9	12.1	12.8	14.0	9.0	14.7	20.3

Appendix II: Grading Scheme for Cribra Orbitalia and Porotic Hyperostosis. From Stuart- Macadam, 1991:109.

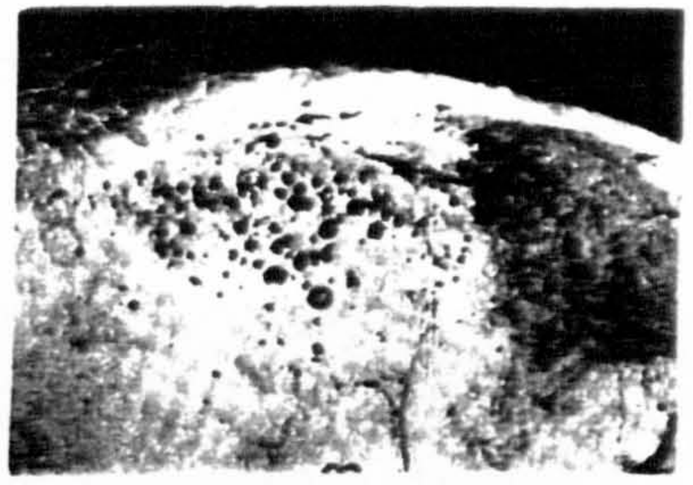
NORMAL

TYPE 1

0 = normal bone surface
1 = capillary-like impressions on the bone
2 = scattered fine foramina
3 = large and small isolated foramina
4 = foramina have linked into a trabecular structure
5 = outgrowth in trabecular form from the outer table surface



TYPE 2



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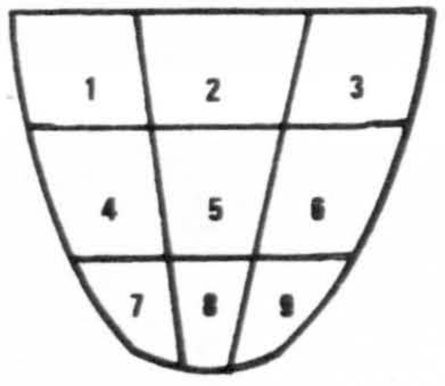
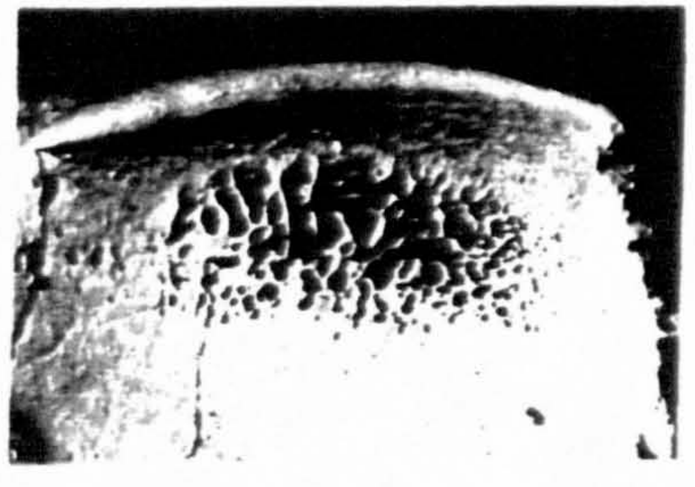




1 = Antero-lateral sector
2 = Antero-intermediate sector
3 = Antero-medial sector
4 = Middle-lateral sector
5 = Middle-intermediate sector
6 = Middle-medial sector
7 = Postero-lateral sector
8 = Postero-intermediate sector
9 = Postero-medial sector

Appendix III: Grading Scheme for Chronic Maxillary Sinusitis.
From Boocock et al., *1995.*

a) White or grey deposits of woven bone.
(under a plaque of lamellar bone)

b) Isolated spicules of lamellar bone

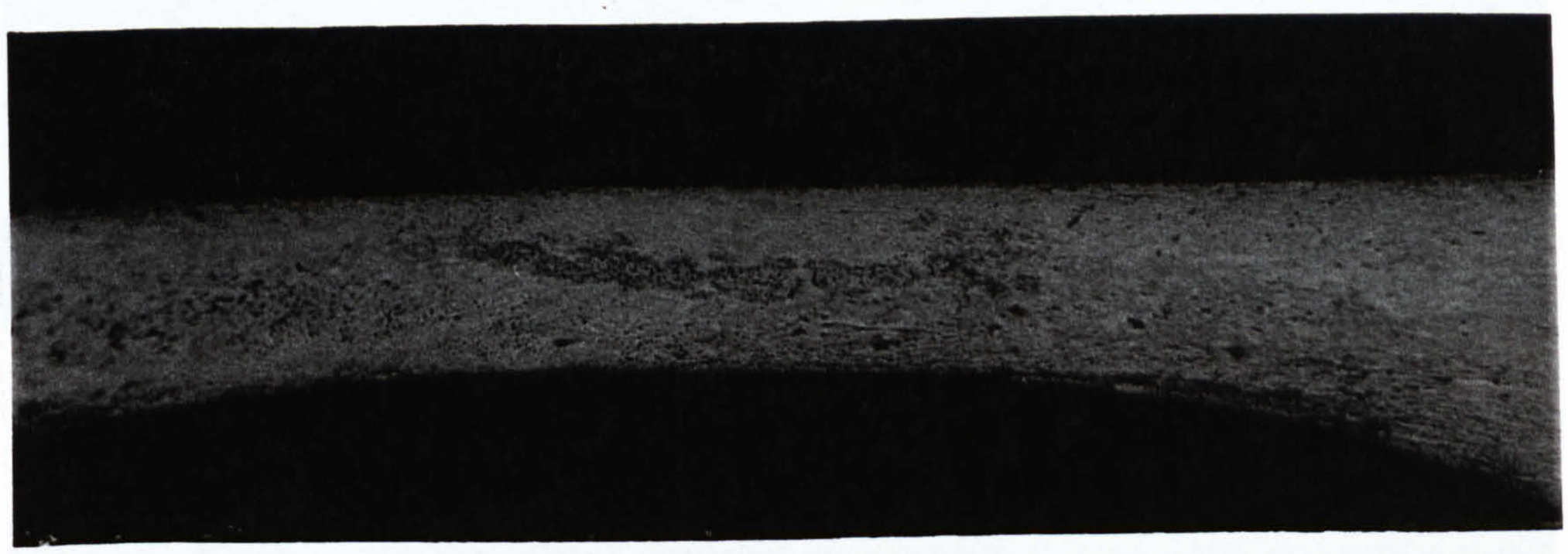
c) Clusters of connected spicules

d) Plaques of lamellar bone

Appendix IV: Grading Scheme for New Bone Formation

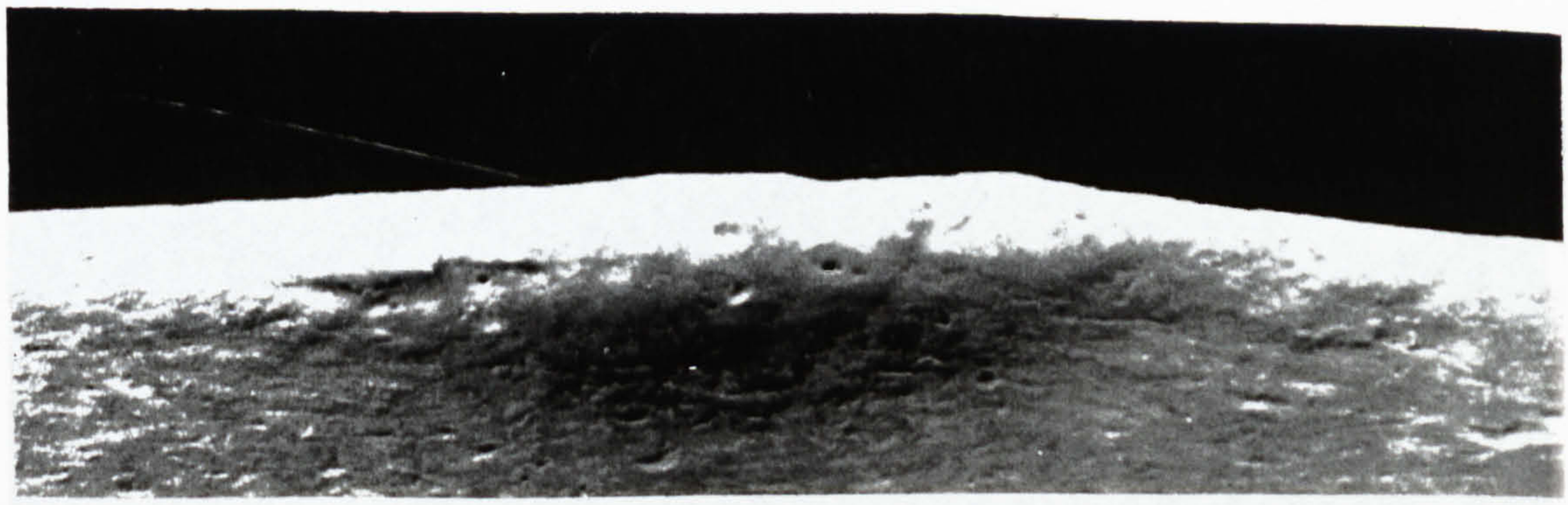
(Specimens from the Calvin Wells Collection, University of Bradford).

1. Inflammatory pitting



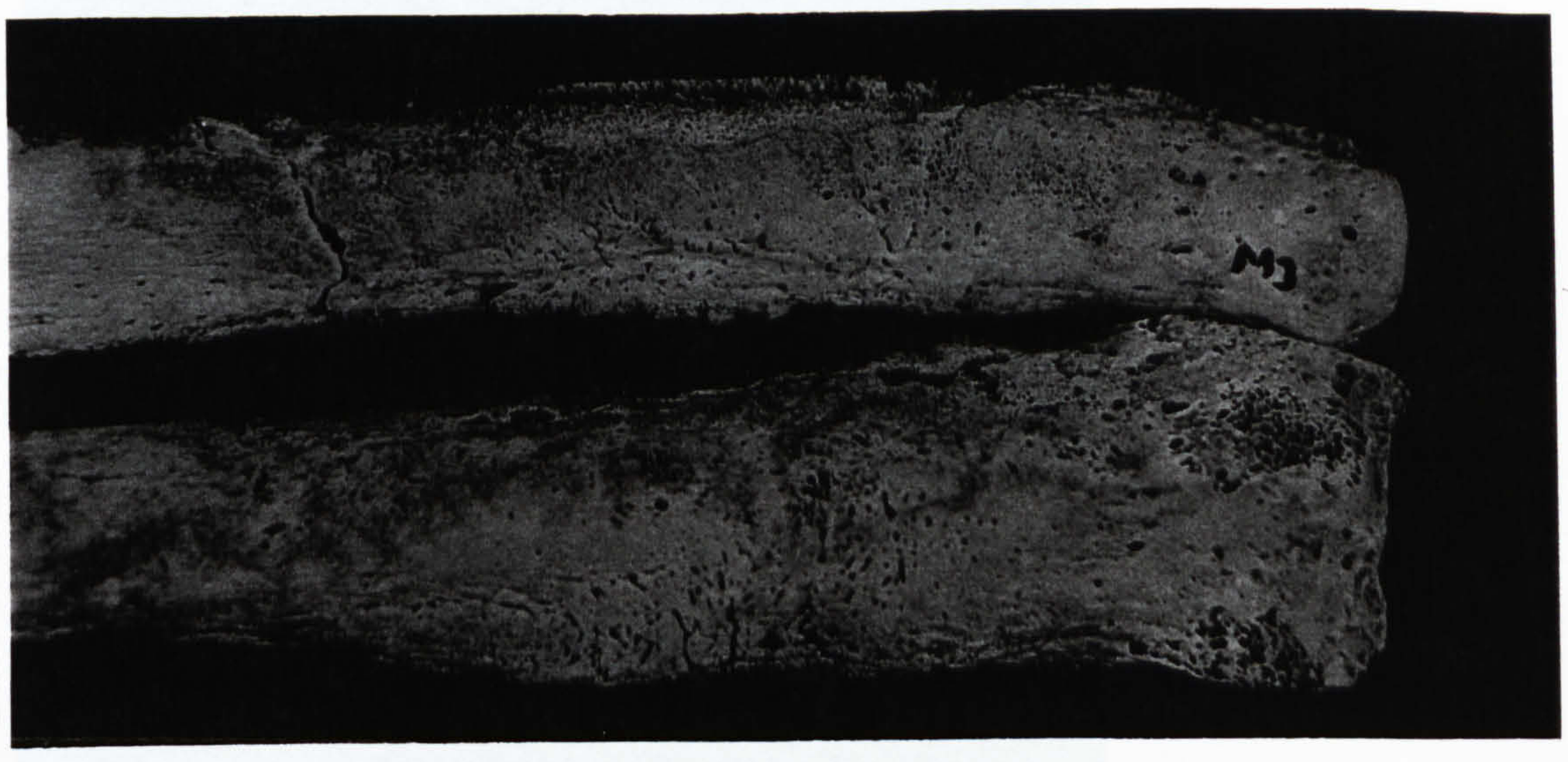
2. Localised deposit of woven bone covering up to one third of the shaf

3. Diffuse woven bone. Covering two thirds of the shaft

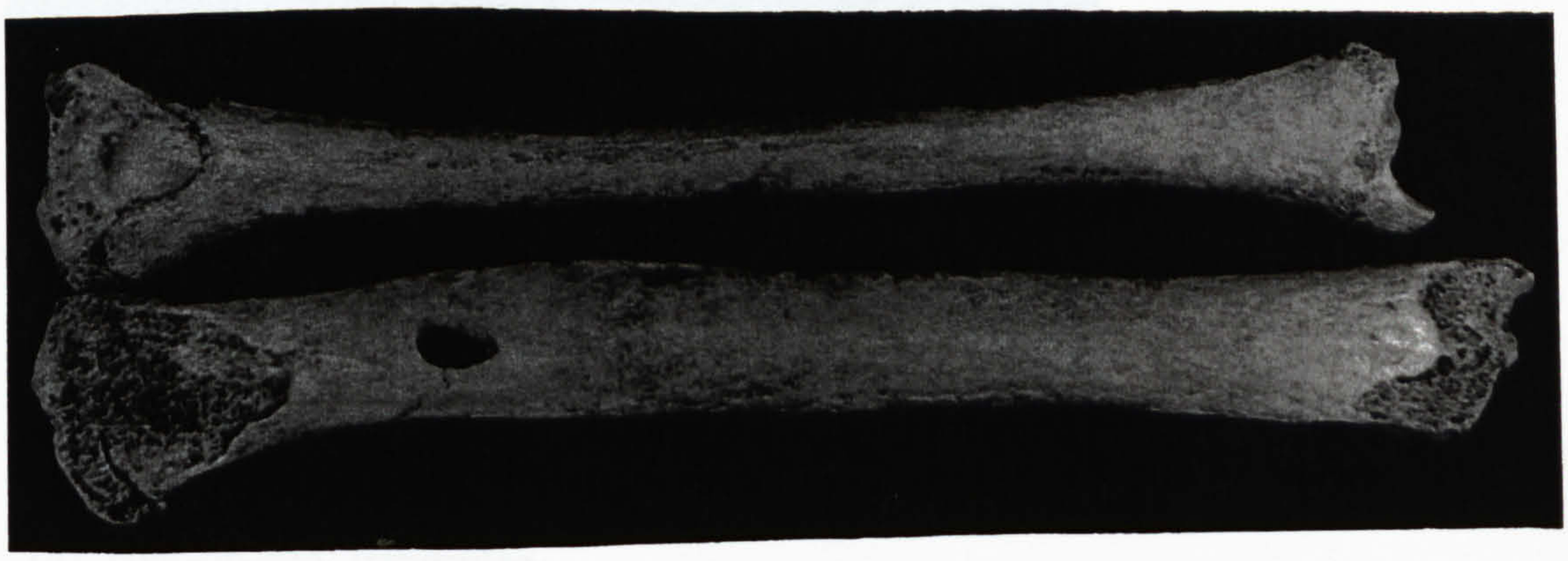


5. Diffuse lamellar bone

6. Mix of lamellar and woven bone



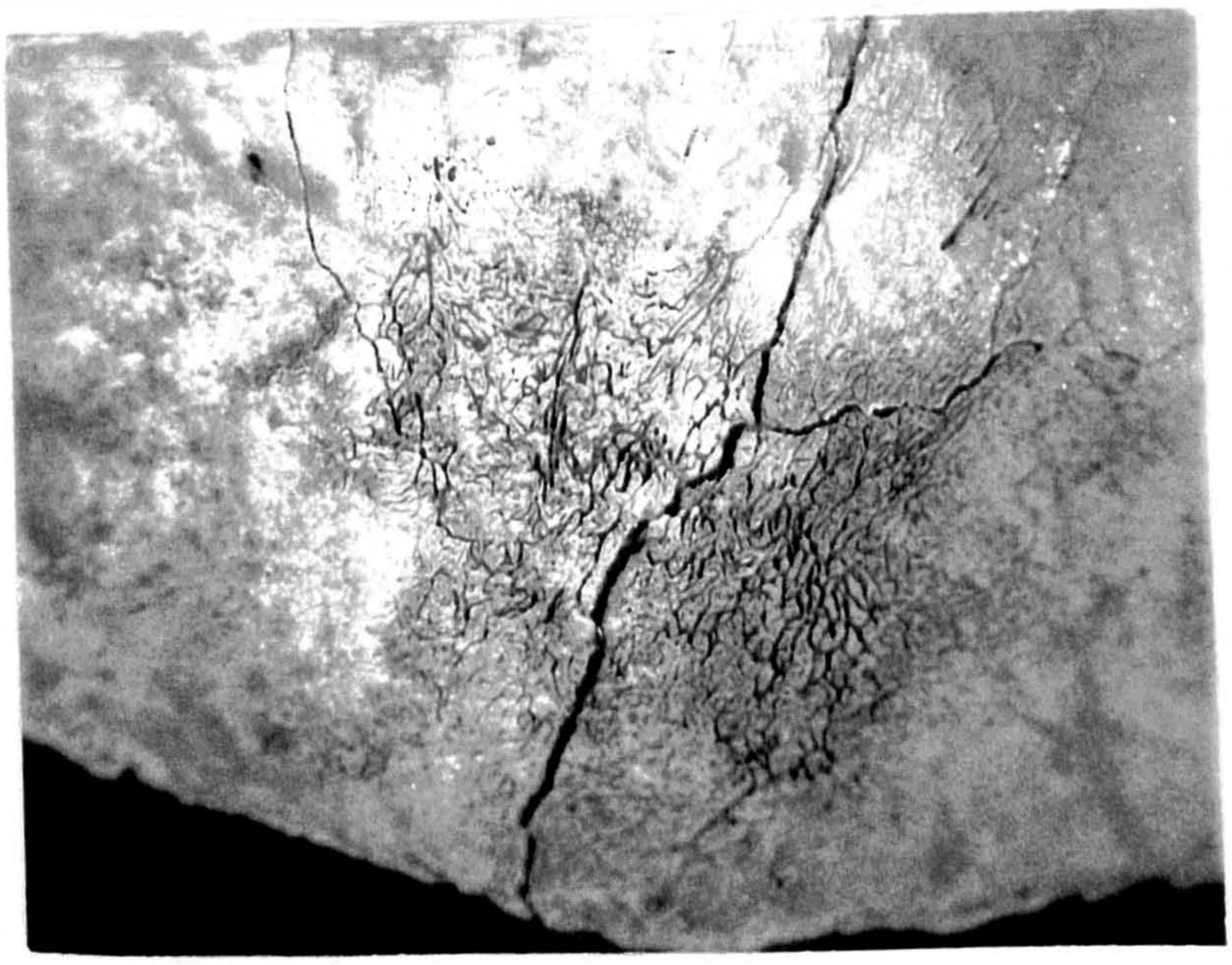
7. Sclerosing osteomyelitis (osteitis)



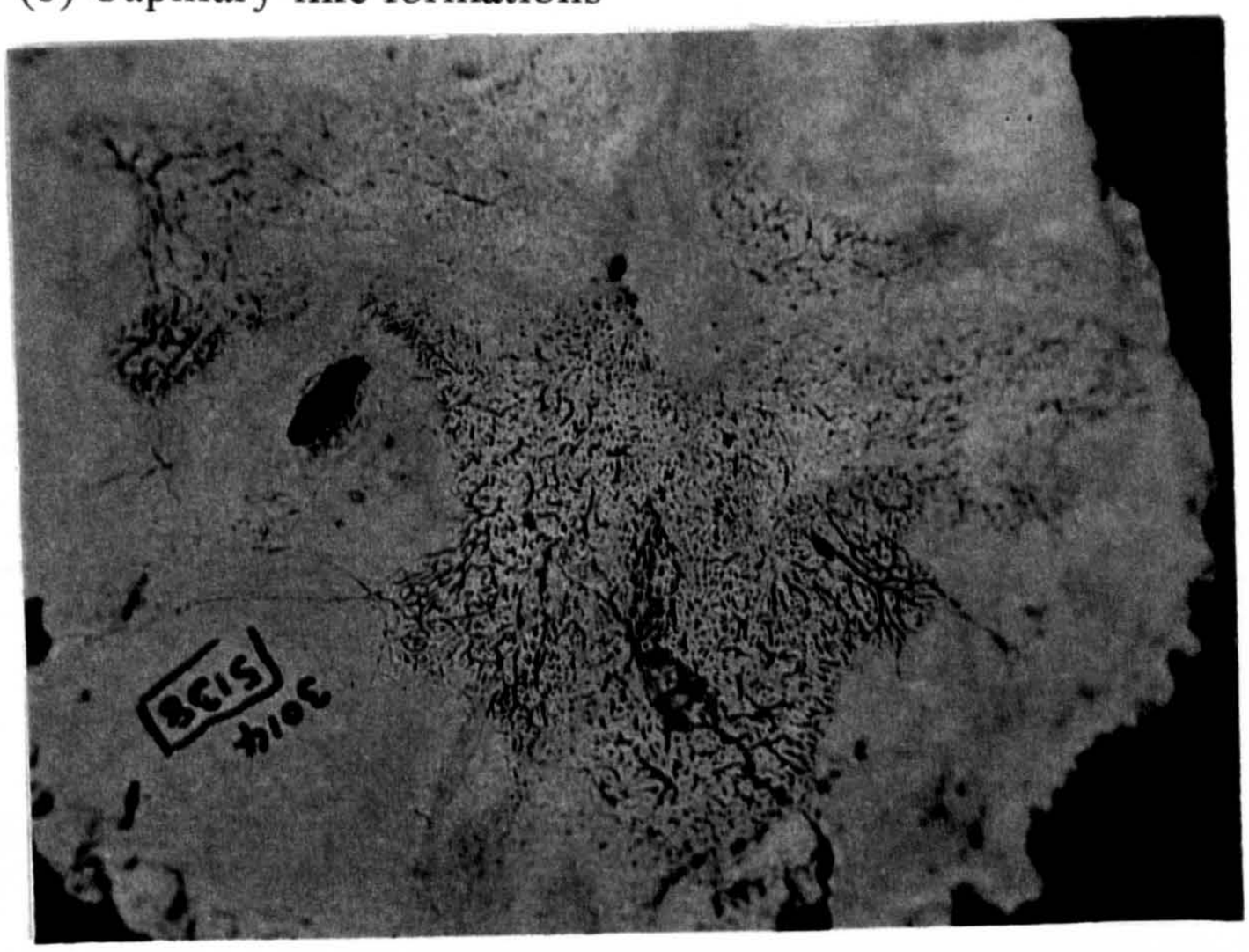
8. Osteomyelitis

Appendix V: The Different Appearances of Endocranial lesions

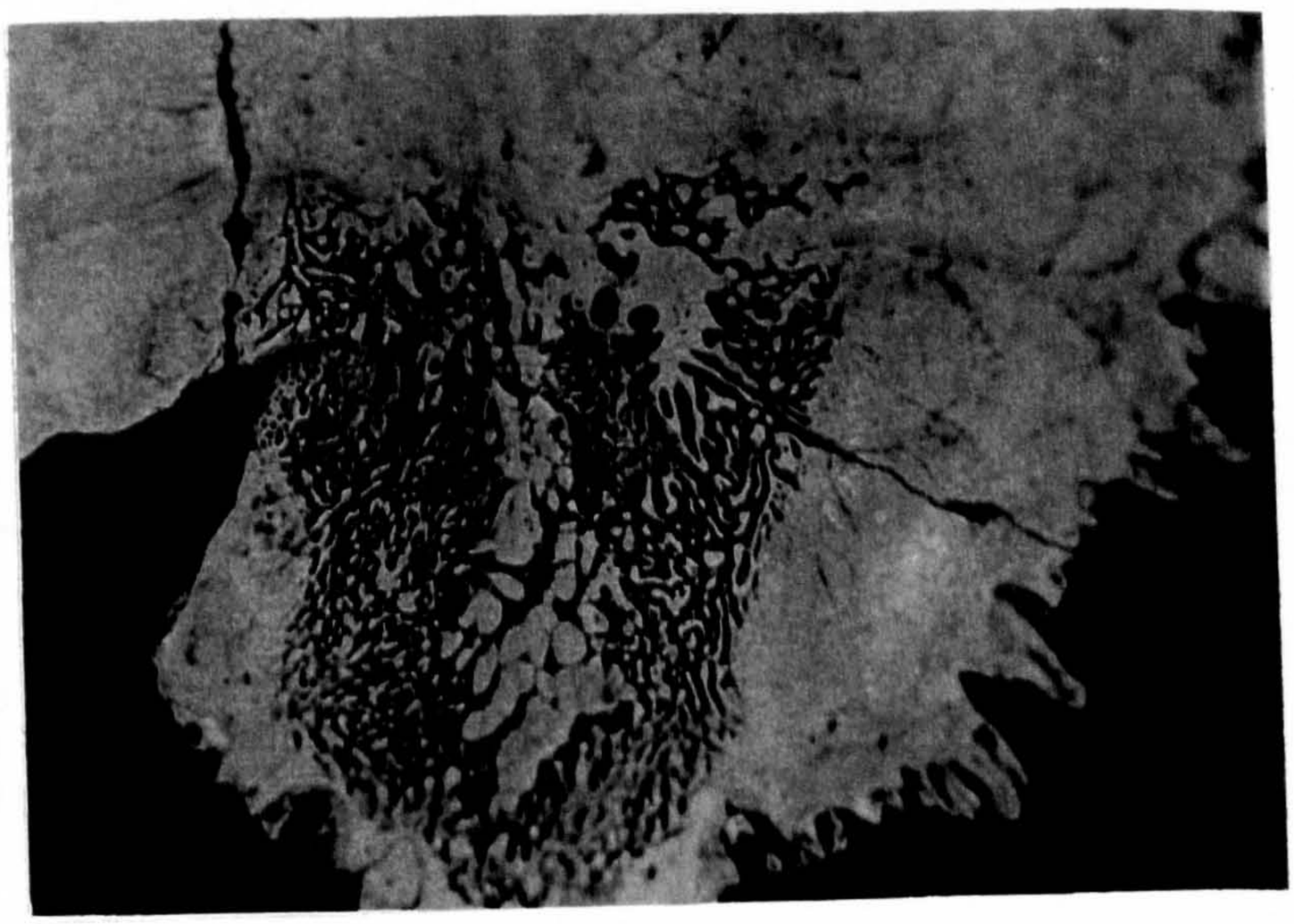
(Specimens from the Calvin Wells Collection, University of Bradford).



(b) Capillary-like formations



(c) Vascular impressions through grey or white woven bone



(d) 'Hair-on-end' trabeculae

Appendix VI: Life Tables

Raunds Furnells

Age	*D(x)*	*d(x)*	*l(x)*	*q(x)*	*q2(x)*	*L(x)*	*T (x)*	*eox*
0-0.5	23	19.30	100.00	0.19	0.38	45.20	430.90	4.30
0.6-2.5	38	32.00	80.70	0.39	0.20	122.90	385.70	4.80
2.6-6.5	25	21.00	48.70	0.43	0.11	148.90	262.80	5.40
6.6-10.5	18	15.10	27.70	0.54	0.14	78.60	113.90	4.10
10.6-14.5	11	9.20	12.60	0.73	0.19	31.20	35.30	2.80
14.6-17.0	4	3.40	3.40	1.00	0.41	4.10	4.10	1.20
Total	119 (of 363)							

St. Helen-on-the-Walls

Age	*D(x)*	*d(x)*	*l(x)*	*q(x)*	*q2(x)*	*L(x)*	*T (x)*	*eox*
0-0.5	10	5.20	100.00	0.05	0.10	48.70	555.70	5.50
0.6-2.5	45	23.40	94.80	0.24	0.13	157.80	507.10	5.30
2.6-6.5	64	33.00	71.40	0.46	0.12	214.10	349.30	4.90
6.6-10.5	49	25.50	38.40	0.66	0.17	100.00	135.17	3.50
10.6-14.5	18	9.30	12.90	0.72	0.18	32.17	35.17	2.70
14.6-17.0	7	3.60	3.60	1.00	0.41	3.00	3.00	0.80
	193 (of 1041)							

Wharram Percy

Age	*D (x)*	*d(x)*	*l(x)*	*q(x)*	*q2(x)*	*L(x)*	*T (x)*	*eox*
0-0.5	46	17.00	100.00	0.17	0.34	45.70	437.90	4.40
0.6-2.5	70	25.80	83.00	0.31	0.16	133.20	392.20	4.70
2.6-6.5	85	31.40	57.20	0.55	0.14	161.80	259.00	4.50
6.6-10.5	46	17.00	25.80	0.65	0.17	67.50	97.20	3.80
10.6-14.5	13	4.80	8.80	0.54	0.14	24.90	29.70	3.40
14.6-17.0	11	4.00	4.00	1.00	0.42	4.80	4.80	1.20
	271 (of 681)							

Christ Church Spitalfields

Age	*D (x)*	*d(x)*	*l(x)*	*q(x)*	*q2(x)*	*L(x)*	*T (x)*	*eox*
0-0.5	40	23.50	100.00	0.23	0.46	44.10	293.70	2.90
0.6-2.5	82	48.20	76.50	0.63	0.33	99.60	249.60	3.30
2.6-6.5	24	14.10	28.30	0.50	0.13	82.90	150.00	5.30
6.6-10.5	12	7.00	14.20	0.49	0.12	41.70	67.10	4.70
10.6-14.5	6	3.60	7.20	0.50	0.13	21.10	25.40	3.50
14.6-17.0	6	3.60	3.60	1.00	0.42	4.30	4.30	1.20
	170 (of 968)							

Bradford, West Yorkshire (1856)

Age	*D(x)*	*d(x)*	*l(x)*	*q(x)*	*q2(x)*	*L(x)*	*T (x)*	*eox*
0-0.5	155	41.66	100.00	0.42	0.84	39.58	178.17	1.78
0.6-2.5	143	38.44	58.34	0.65	0.34	74.32	138.59	2.37
2.6-6.5	60	16.13	19.90	0.81	0.20	46.16	64.27	3.23
6.6-10.5	7	1.88	3.77	0.49	0.12	11.03	18.11	4.80
10.6-14.5	3	0.81	1.89	0.42	0.11	5.79	7.08	3.74
14.6-17.0	4	1.08	1.08	1.00	0.42	1.29	1.29	1.19
	372 (of 565)							

Bradford, West Yorkshire (1931).

Age	*D(x)*	*d(x)*	*l(x)*	*q(x)*	*q2(x)*	*L(x)*	*T (x)*	*eox*
0-0.5	41	59.42	100.00	0.59	1.18	35.14	277.15	2.77
0.6-2.5	13	18.84	40.58	0.46	0.24	59.20	242.01	5.96
2.6-6.5	3	4.35	21.74	0.20	0.05	76.30	182.81	8.40
6.6-10.5	4	5.80	17.39	0.33	0.08	56.51	106.51	6.12
10.6-14.5	2	2.89	11.59	0.25	0.06	39.56	50.00	4.31
14.6-17.0	6	8.70	8.70	1.00	0.41	10.44	10.44	1.20
	69 (of 442)							

Appendix VII: Long Bone Measurements (mm) (weeks and years)

Raunds Furneus

	Mean age	*Humerus*	*Radius*	*Ulna*	*Femur*	*Tibia*
5330	33.0			48.6	61.2	
5294	34 4		46.6		63.2	54.6
5301	35 8	60			66 8	
5293	35.9	59.9	49		65.9	58.7
5245	36 0	64.5				
5267	36 0			55.5	70 1	60.2
5305	36 0	60.9			68.7	60.6
5334	36 0	58.2				60
5351	36.0				39.9	
5196	36.5	62.1	49 6	56.1	68.9	59.9
5276	37.3	63.8		58.3	71.1	61.6
5198	38.0	58.5	47.8	53.9	68.4	58.9
5251	38.0	63.9	51.7	58 6		65.4
5327	38.4				75.3	
5279	38.6	65.4	52.7	61.8	73.2	64.1
5277	38.7	64.5	51.2	59.2	77	67.1
5319	38.7	65.7				
5355	38.7	64.7	61.2	57.9	72.3	62
5034	38.9	66.5	52.8		77.6	65.3
5141	39.0	67.4	54.6			
5300	39.0	66 6	56.5		76.3	
5060	39.8	66		61.7	76 8	69.8
5350	39.8	68.2	55.6	62.9	79 1	
5185	0.3		73.1	81.3		
5101	0.4	73.5	56.5	64.7	90	
5169	0 4	76 8	62.4	70 6	89 9	
5309	0.4	99				
5140	0.6		72 8		110.6	
5096	0.7	96.4	76.3	85	118.9	
5210	1.0	116	90	101	136	109
5258	1.0	105			125	104
5004	1.1	110 2	81.9	90		
5310	1.3		77		131	106
5011	1.4		87	96.4		117.1
5109	1.4	112	85	92	141	114
5302	1.4	110	85		140	
5356	1.6			95.2		
5264	1.7	141	104		173	136
5292	1.7	130	94		167	135
5012	2.2				153	122
5322	2.2	131.7				
5208	2.3	123.3			160.8	127.7
5354	2.4			104.5		
5023	2.5	143	98	109	190	148
5238	2.5	148				
5271	2.5	145	105	117	190	
5345	2.5	133	95	108	172	135
5174	2.6	130		109	179	137
5005	2.7		100		187	143
5274	2.8	142	101	116	187	157
5280	2.8	126	95	107	171	134
5212	2.9	145	110	122	193	154
5199	4.1	144		114	189	145
5349	4 2	136	102	112	172	
5343	4.4	151				
5303	4.6					193
5131	4 8	158	122	135	213	173
5297	4 8	162	124	140		174
5032	5.3	161	120	130	204	172
5088	5.4			128.7		
5084	5.6				233	
5335	6.1	162	119	132	213.3	162
5346	6.3				248	200
5249	6.4	181			247	
5290	6.4	190				
5041	6.5	191	145	161	256	212
5116	6.9	188	142	159	270	
5194	7.3	146	104	115	189	152
5216	7.3	192	137	153	269	214
5163	7.4				227	
5014	7.6	214	150	162	294	240
5075	7.9	224	167	187	303	254
5265	7.9	202	151	167	272	
5125	8.1	173				
5281	8.3	198	154	169	283	224
5317	9.2	238		185	321	250
5065	9.5	216	154	173		
5215	9.9				290	
5138	10.7	227	173	193	326	254
5200	11.3	280	188	208	367	291
5348	11.9	221	161	182	321	
5030	12.1	267				
5037	12.2	251	182	203	349	275
5324	12.3	249			361	278
5168	12.7	229	165		324	255
5013	14 6	248				273
5311	15.1	238	175	198	340	
5059	15.4	270		212	368	325
5352	15.4	252	186	216	334	

St. Helen-on-the-Walls

	Mean age	*Humerus*	*Radius*	*Ulna*	*Femur*	*Tibia*
5630b	37.0	61.8				
5825	38 0	65				
5826	38.0				74.5	
6012a	38 0	65	52.5	59.9	75.4	
5454	38 6	66.3	53	59.8		
5026	38.8	65.8				
5092	39 0		53.4		77.2	66.5
5080	0.3	82.8	64.7		99.3	83.6
5183	0.3					86.1
5660	0.3	78.4		73 4		
5072	0.9	103.5				
5446	0.9	101.7			129.94	107.9
5276	1.0	106	77.7	85 3	135.2	104.7
5391	1.1					102.8
5360	1.2	93.8				
5469	1.3				174	140
5272a	1.3	126	92	102	162	127
5282	1.4		82.9	90.6	136.3	
5274	1.5	130		111	160	135
5765	1.5				165	
5696	1.8	115.3			147.5	118.57
5154	2.1	134			181	
5834	2.1	132.3		89.4	167.5	
5352	2.2	124.9				
5383	2.5	133.5	101.1			
5003	2.7	141			195	156
5947	2.7	122			167	131
5698	2.8	143 9	105.5	111.8	177.1	142.4
5777	2.8	137			171	140
5353	3.4	152		123		
5387	3.5	148	110	120	200	
5880	3.9	144			186	
5689	4.5	164				168
5084	4.6	181	135			203
5946a	4.8		149	161	278	221
5976	4 9	155	111	122	196	156
5133	5.2	183			235	190
5286	5.2	165	116	131	216	168
5757	5.3	169	116	126		
6079	5 4	176	124	136	228	183
5128	5.5	165		131	213	171
5513	5.6	171	124			
5476	5.7	163			232	188
5752	5.7	165	118	131	222	
5089a	5.8	163			220	174
5077	6.0	170	123	138	227	
5628	6.1	178	126	140	246	
5682	6.1	193		144	254	
5675	6.2			159	286	
5484	6 5	184	131	140		
5085	6.6				276	230
6086	6.6	183	130	146	243	199
5778	7.1			168	260	213
5137	7.2	201	143	155	289	
5467	7.3	187	140	155	251	202
6023	7.7	185	132	145	275	214
5697	7.8	215	154		283	234
5793	7.8		140	153	265	
5795	8.5			175	305	234
6092	8.7	205				
5634	8.8	147	133	191		
6034	9.1	229	171	190	325	252
5904	9.2	192			278	213
6080	9.4	224			315	
5444	9.5	214	153	173	307	226
5873	9.5	212	152	172	291	235
5771	9.7	185		153		208
5510	9.8		190			274
5138	9.9	233		182		
5669	10.1	233	170	187	313	251
5943	10 6	229	175		304	248
6004	10.6	249			327	265
5599	10.7	240	171			
5121	11.4	266			357	
5189	12.1		202		379	290
5695	12.1	228	168		320	262
5252	12.3	260		232	386	318
5526	13.6	235	210		407	333

Wharram Percy

	Mean age	*Humerus*	*Radius*	*Ulna*	*Femur*	*Tibia*
NA121a	29.0	44.8		41.8		
NA142a	29.8			42.3	49 5	44.8
G457	31.0	47.4			54.9	47.1
NA56a	31.3	50 0			53.6	
G476	32.9	53.1	43 0	49.2	57.9	52.3
NA124	33.1			49.9		
NA6a	33 2	54 3		49 6	59 3	
NA161	33 3	54.3	43.2	49.0	58 9	54 4
NA233a	33.6	54 6				
NA61	34.2	55.3	44.7	51.3	63 0	56 0
NA60a	34 5	56.1		53.0		
NA82	34 8	58 3		51.8		56.2
EE59	35.0	57.5				
NA62	35.0	58.4		53.1	65.1	
NA99	35 0	59 6	46.1	52.7	66.2	57.9
NA67	35 2	57 8	46.9	52.1		59.2
V1	35.5	57.4	46 8	54 5	68 7	58 9
NA129	35.7	59.2				
V3	35.7	60.5	47.5	53.9	68 0	57.9
NA32	35.8		48 8	54.3	65.7	59.4
V63	36.0				68.4	
NA42	36.4	60 4	49.2			
EE88	36 9	61.5		57.3	70 9	62 8
EE34	37.0		48 9	55 2	67.3	60 5
NA142	37.0	64.2			71.7	61 1
NA4a	37.9	64 6	51.4	59.3		
NA37a	37.9	64.5		59.4	75 0	62.0
NA76	38.0				75.6	62.3
G320	39 0	65.0	54 0	62.3	72.7	
WCO125a	39 0	68 5			76.4	
NA149b	39.5		52.9		79.9	68 1
G470	0.3	74 8	61.9		87.7	74 8
G534	0.3	84.7			102.7	87.0
G686	0.3	79.3	60 8			
NA60	0.3	78.3				
NA106	0.3	78.6	61.5	69 6		
NA139	0.3	76.9			90.9	
NA209	0.3	71 8				
NA221	0.3	80.4	65.3	74 0	94.0	
NA232	0.3	74.4	59.4	66.7		73.2
WCO104	0.3	85.0		76 1		
EE53	0.4	81 3	65 5	73.5	98 8	
NA43	0 4	78 0			92.2	79.8
NA85	0 4	79.3				
NA150	0.4	83 8				
NA191	0.4	82.0	65 2	70.4	96.0	85.0
NA92	0.5	83.2			97.2	83 6
NA119	0.5	103 0			130.0	
WCO42	0.5					76.7
NA103a	0 6	100 0		86 0	120.0	102 0
NA108	0.6					89.8
NA194	0.6		60.1			
EE52	0.7	91 0			91 4	
NA119	0.7	103.0			130.0	
EE68	0 8		78.7			108.4
NA130	0.8					122.7
NA216	0 8		74 3			
V10	0 8	93.1				93 8
WCO85	0.8				122.0	
NA37	0 9	114 0	82.4		144 0	113.9
NA50	0 9	104 0			129 0	
G522	1.1	104.2	72 8	82.6	119 5	103.7
NA29	1.1	97.3	75 1	81 9	118.4	98 5
NA107	1.1	91.9			112.2	
NA189	1.1	105 0	85 0		130 0	113.0
NA118	1.2		95.0	103 0		
G655	1.3	115 6				
WCO115a	1.3			89.4	133 8	108 8
NA36	1.4	103.0				
NA164	1.4		76 8	85 8	134.0	105.0
G298	1.5			89.5		
G430	1.5	112.0	81.7	90 6	141 0	111 0
SA153	1.5	102.0				
NA70	1.6	125 0			160.0	145 0
WCO72	1.6	139.2	98.2			126 8
WCO108	1 8	132.0			160.0	127 8
G364	1.9			108 0	175.0	142.0
G363	2 0	127.0			162.0	125.0
G697	2.2	122 0	89.0	100 0	153 0	125.0
NA221	2.2	148.0			194.0	
SA55	2.3	120.0	95 0	100.0	151.0	125.0
NA116	2.6	122.0	90.0	99 0		123 0
EE73	2.7	127.0		102.0	165.0	130.0
NA184	2.7	129.0				
V22	2.7			120.0		162.0
V25	2.7	148 0	107.0	118 0	198 0	156.0
WCO72	2.7		110.0	120 0	200.0	
EE17	3.0					152.0

Christ Church Spitalfields

	Mean age	*Humerus*	*Radius*	*Ulna*	*Femur*	*Tibia*
2065	30 0	48	38	42		47
2222	32.0	51	42	48		49
2080	33.0	52	42	49	61	
2968	34.0				63	
2631	36 0					60
2906	36 0	61			67	60
2648	37.0	62	49			
2794	37.0	62	50	58	72	
2862	37.0	62	51	59		
2836	37.5	64	50	57	74	63
2797	38 0		54	61.5	72	63
2834	38.0	65	53	59	74	63
2250	39.0	67	52	60	77	66
2732	39.0	67	54	63	78	
2833	39.0	69	54	61	78	67
2242	39.5	67	54	61	78	69
2247	0.3	70	58	65	79	70
2561	0.3	88	66	74		
2630	0.3	79	61	68	92	80
2659	0.3	71	56	63	84	71
2774	0.3	71			83	71
2780	0.3		52		82	69
2786	0.3	74	57	64	87	75
2844	0.3	71	56	64		75
2270	0.4	73.5	55	62	89	73
2277	0.4	76	58		93	79
2364	0.4					87
2478	0.4	61	48	54	71	61
2529	0.4		58	67		
2657	0.4	77	61.5	67	91	80
2736	0 4	69	57	64	83	72.5
2763	0 4	97			122	104
2775	0.4	91	75	81	112	97
2147	0.5		60	66	97	84
2315	0.5		62	69	94	82
2453	0.5					87
2482	0.5		69			94
2512	0.5		60	67	91.5	76
2768	0.5	68	55	61		67
2813	0.5	83		67	104	84
2261	0 6	80	61.5	70	104	85
2555	0 6	91	68	76	116	95
2316	0.7	82	64	71	104	90
2412	0 7	89	68	72	111	92.5
2491	0.7			81	111	99
2530	0.7	101	76	85	122 5	106
2480	0 8				121	
2506	0.8	98	76		122	
2724	0.8	91	68			92
2737	0 8	100		79	127	103
2793	0 8	85	64	73	105	88
2947	0.8		70			
2505	0.9	104			131	106
2531	0 9			75		92
2876	0.9	80 5	62	68	102	85
2878	0 9	87	64	71	110	89
2452	1.1	98				
2533	1 1	96			124	
2148	1.2	97	74	77	122	105
2373	1.2	95			122	102
2532	1.2	98	76	79		
2673	1.2	110	82	89	144	114
2691	1.2	84 5	76			
2757	1.2	120	85	93	158	127
2206	1 3	112	80	88		121
2282	1.3	103	77	82.5	126	103
2734	1.3	93	68	77	116	94
2771	1.3	109	80	90	143 5	133 5
2303	1.4	119	91	100	152	125
2429	1.4	99	75			
2431	1.4	121	91	98	156	128
2441	1.4	116	88	97	143	125
2578	1.4	104			133	107
2582	1 4	104	79	84	134	108
2792	1.4	92	72	78	117	94
2905	1.4	114	84	92	149	123
2626	1.5	102			134	109
2779	1.5	126	100		164	129
2248	1.6	119	86	92	152	128
2772	1 7	93			116	98.5
2514	1.8		104			
2656	1 8	97	71	77	123	97.5
2420	1 9	119	89			
2450	2 0					137
2456	2.0	131				
2562	2.0	124.5				
2815	2 0	104	75	81.5	134	111
2520	2 1				179	144

Wharram Percy continued...

	Mean age	Humerus	Radius	Ulna	Femur	Tibia
G576	3.2	138.0	104.0		180.0	148.0
G281	3.5	140.0				
G422	3.5	153.0	114.0	128 0	205.0	161.0
G467	3.5	133 0	97.6	109.5		
EE71	4.3	149 0	114.0	122.0	201.0	
EE90	4.5	147.0	114.0	125.0	190.0	
G314	4.5	128 0	119 0			
G542	4.5	170.0			229.0	180.0
G614	4.5	155.0	116.0	127.0	212 0	168.0
G683	4.5	154.0				
NA120	4 5	155.0			210 0	173.0
WCO3	4 5	163.0		130.0	210.0	170.0
WCO27	4.5	155.0			215.0	
NA201	4.6	153.0	110.0	123.0	196.0	162 0
NA35	5 0	172.0	123 0	139.0	232.0	190.0
EE55	5.1	160.0				
G424	5.1	175.0	130.0	142.0	234.0	185.0
G433	5.1	171.0	121.0		228.0	179.0
NA24	5.1		102.0		177.0	137.0
NA30	5.1	159.0	116 0	130.0	220 0	173.0
NA58	5.1	142.0	106.0	121.0	204.0	166.0
NA122	5.1	162.0		130.0		
V49	5.1	161.0				
EE42	5 2	175.0		139.0		
NA110	5.2	144.0	101.0		187.0	151.0
G304	5.3		132.0	142.0		
NA48	5.3		113.0	125 0		
NA98	5 4	160.0	116 0	130.0		171.0
V23	5.5	173.0				
EE74	5.7	152.0		126.0	201.0	
G558	5.7	170.0	131.0	142.0	230.0	181.0
CN4	5.8	201.0	133.0		272.0	210.0
EE76	5.8	169 0	120.0	133.0		
G510	6.0	178.0	134.0	147.0	240 0	195.0
NA23	6.1	175.0	127.0	140 0	233.0	185.0
NA111	6.3	173.0	137.0	150.0	250.0	196.0
EE102	6.4	193.0	142.0	152.0	260.0	
NA135	6 4	163.0	122.0	138.0		
EE65	6.5	181.0	130.0	144.0	250.0	193.0
EE66	6.5	200.0	147.0		277.0	
EE75	6.5	192.0		148.0		
NA65	6.5	198 0	151.0	165.0	276 0	222.0
NA175	6.5	184 0	138 0			
WCO202	6.5	181.0	133.0	146.0	240.0	185.0
WCO52	6 9	192.0				
NA19	7.0	206.0			285.0	
WCO99	7.3	189.0	130.0	144.0	255.0	192 0
G500	7.5	194.0		165.0	270.0	222.0
G506	7.5	190.0	141.0	157.0		
WCO140	8.2	205.0	135.0	155.0	273.0	217.0
WCO164	8.4	204.0	146.0	160.0	275.0	
G708	9.0				315.0	255 0
G658	9.1	197.0	152.0		280.0	215.0
G456	9.3	240.0			330.0	280.0
NA2	9.9	236.0	168.0	185.0	321.0	
NA174	9.9	225 0	149.0	168.0	295.0	223.0
WCO5	9 9	208.0		171.0		240.0
WCO82	9 9	215.0	160.0			234.0
NA198	10.3	225 0	161.0	182 0	314.0	248.0
NA138	10.5	233.0	167.0	182 0	312.0	246.0
WCO6	11.3	214.0				
V7	11.4				347.0	271.0
WCO69	11.4		170.0		327.0	248.0
WCO103	12.5				348 0	286 0
G497	12.7	253.0	194.0	215.0	340.0	280.0
NA105	12.7	243.0	183.0	198.0	334.0	268.0
WCO79	12.7	263.0	191.0	205.0	362.0	273.0
WCO186	12.7	230.0		192.0		
G689	14.6		202.0		385.0	300.0
V24	14.6	247.0	179.0	199.0	350.0	270.0
NA4	16.4			242.0	399.0	
WCO92	16.9		200.0			297.0

Christ Church Spitalfields continued...

	Mean age	Humerus	Radius	Ulna	Femur	Tibia
2845	2.3		91	99	161	
2639	2 7	148	109		199	163
2644	2.7	126	90	96	159	124
2658	2.7	150	108	118	208	166
2702	2.7	127	94	103	170	138
2723	2.7	139			196	
2826	2.7	109.0	82	90	142	113
2660	3.2	150	105	116	195	160
2625	3.5	146	106	115	194	155
2668	3.5	146	105	114	193	154
2758	3.5	141	105	115	194	
2264	3.9	149	105	114	200	163
2621	3.9	134	95	99	180	141
2179	4 5	162	119	127	216	172
2730	5.1	138	97	106	184	141
2199	5.2	132	102	113	191	165
2672	5.5	151	112	122	207	163
2823	5 6	173	129	139		
2583	5.8	167	119	130	228	183
2692	6 0	156	113	124	216	166
2128	6.1	172	124	136	238	197
2866	6.6	164	117	133	226	179
2235	6.7	214	154	170	297	242
2009	6.9	184	131	146	263	208
2266	6.9	161	116	130	224	181
2145	9.1	209	149	160	321	252
2662	9.3	221	172	190		266
2280	9.5	201	147	160	289	226
2677	9.5	215	153	164	313	241
2139	9.8	226	169	181	321	271
2135	10.3	223	153	166		253
2239	11.3	217	162	178	303	240
2200	11.3	224			306	257
2107	11.5			194	360	285
2721	12.4		182	196	365	297
2755	12.8	266			371	296
2217	15.4		184	196	373	300

Appendix VIII: Number and Percentage of Indicators of Stress

Cribra Orbitalia

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with orbits	%a (N)	%b	number	with orbits	%a (N)	%b	number	with orbits	%a (N)	%b	number	with orbits	%a (N)	%b
0-0.5	25	13	2 (2)	15	10	4	0 (0)	0	45	34	1 (2)	6	40	17	5 (6)	35
0.6-2.5	36	24	13 (11)	46	45	21	13 (11)	52	70	55	17 (34)	62	82	54	26 (29)	54
2.6-6.5	25	20	18 (15)	75	64	23	19.5 (17)	74	85	67	23.5 (47)	70	24	23	15 (17)	74
6.6-10.5	18	14	12 (10)	71	49	30	15 (13)	43	46	26	9 (18)	69	12	12	7 (8)	67
10.6-14.5	11	9	8 (7)	78	18	9	9 (8)	89	13	10	2.5 (5)	50	6	3	3 (3)	100
14.6-17.0	4	4	1 (1)	25	7	0	0 (0)	0	11	8	3 (6)	75	6	2	0 (0)	0
Total	119	84	55 (46)		193	87	56 (49)		301	200	56 (112)		170	111	57 (63)	

%a-percentage of total individuals with orbits; %b-percentage of individuals with orbits in each age group.

Enamel Hypoplasias

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with teeth	%a (N)	%b	number	with teeth	%a (N)	%b	number	with teeth	%a (N)	%b	number	with teeth	%a (N)	%b
0-0.5	25	3	0 (0)	0	10	3	0 (0)	0	45	0	0 (0)	0	40	4	0 (0)	0
0.6-2.5	36	21	0 (0)	0	45	24	1 (1)	5	70	28	2.5 (3)	11	82	50	5 (10)	20
2.6-6.5	25	19	8 (6)	32	64	26	6.5 (6)	23	85	53	11 (13)	24.5	24	20	3 (3)	15
6.6-10.5	18	15	10 (7)	47	49	26	16 (15)	58	46	24	8 (10)	42	12	11	7 (6)	54
10.6-14.5	11	10	8 (6)	60	18	13	10 (9)	75	13	8	6 (7)	87.5	6	3	2 (2)	67
14.6-17.0	4	4	5.5 (4)	100	7	0	0(0)	0	11	7	2.5 (3)	43	6	3	1 (1)	33
Total	119	72	32 (23)		193	92	34 (31)		301	120	30 (36)		170	91	24 (22)	

%a-percentage of total individuals with teeth; %b-percentage of individuals with teeth in each age group.

Endocranial Lesions

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with skull	%a (N)	%b	number	with skull	%a (N)	%b	number	with skull	%a (N)	%b	number	with skull	%a (N)	%b
0-0.5	25	20	5 (5)	25	10	6	2 (2)	33	45	36	2 (5)	14	40	17	0 (0)	0
0.6-2.5	36	29	7 (7)	24	45	22	4 (4)	18	70	62	7 (16)	26	82	51	3 (4)	8
2.6-6.5	25	22	1 (1)	4.5	64	31	2 (2)	3	85	72	5 (10)	14	24	21	0.8 (1)	5
6.6-10.5	18	17	0 (0)	0	49	21	2 (2)	9.5	46	29	0.9 (2)	7	12	12	0 (0)	0
10.6-14.5	11	10	1 (1)	10	18	10	1 (1)	11	13	10	0 (0)	0	6	3	0 (0)	0
14.6-17.0	4	4	0 (0)	0	7	2	0 (0)	0	11	8	0 (0)	0	6	2	0 (0)	0
Total	119	102	14 (14)		193	92	12 (11)		301	217	15 (33)		170	117	4 (5)	

%a-percentage of total individuals with a skull, %b-percentage of individuals with a skull in each age group

New Bone Formation

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b
0-0.5	25	18	1 (1)	5.5	10	7	0 (0)	0	45	24	1 (2)	8	40	35	0 (0)	0
0.6-2.5	36	19	0 (0)	0	45	17	4 (4)	23	70	39	3 (5)	13	82	69	1 (2)	3
2.6-6.5	25	21	6 (5)	24	64	34	4 (4)	12	85	53	2 (3)	6	24	22	1 (2)	9
6.6-10.5	18	12	4 (3)	25	49	33	9 (9)	27	46	36	6 (10)	28	12	11	0.7 (1)	9
10.6-14.5	11	8	5 (4)	50	18	9	3 (3)	33	13	8	0 (0)	0	6	6	0.7 (1)	17
14.6-17.0	4	4	2 (2)	50	7	4	1 (1)	25	11	6	0.6 (1)	17	6	4	0 (0)	0
Total	119	81	18 (15)		193	104	20 (21)		301	166	13 (21)		170	147	4 (6)	

%a-percentage of total individuals with tibiae; %b-percentage of individuals with tibiae in each age group.

Dental Disease

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with teeth	%a (N)	%b	number	with teeth	%a (N)	%b	number	with teeth	%a (N)	%b	number	with teeth	%a (N)	%b
0-0.5	25	3	0 (0)	0	10	1	0 (0)	0	45	3	0 (0)	0	40	8	0 (0)	0
0.6-2.5	36	26	0 (0)	0	45	20	1 (1)	5	70	41	0.6 (1)	0.2	82	51	9 (9)	18
2.6-6.5	25	20	4 (3)	15	64	29	7 (6)	21	85	67	5 (8)	12	24	21	7 (7)	33
6.6-10.5	18	15	0 (0)	0	49	20	8.5 (7)	35	46	29	0.6 (1)	3	12	12	6 (6)	50
10.6-14.5	11	9	1 (1)	11	18	13	5 (4)	31	13	13	1 (2)	15	6	4	3 (3)	75
14.6-17.0	4	3	1 (1)	33	7	0	0 (0)	0	11	9	1 (1)	22	6	3	3 (3)	100
Total	119	76	6.5 (5)		193	83	22 (18)		301	162	9 (14)		170	99	28 (28)	

%a-percentage of total individuals with teeth; %b-percentage of individuals with teeth in each age group.

Maxillary Sinusitis

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with sinus [?]	%a (N)	%b	number	with sinus [?]	%a (N)	%b	number	with sinus [?]	%a (N)	%b	number	with sinus [?]	%a (N)	%b
0-0.5	23	-	-	-	10	-	-	-	45	-	-	-	40	-	-	-
0.6-2.5	25	12 [0]	0 (0)	0	45	6 [0]	0 (0)	0	70	8 [0]	1 (1)	12.5	82	21[6]	0 (0)	0
2.6-6.5	36	1 [0]	5 (1)	20	64	11 [3]	3 (1)	9	85	46 [11]	3 (3)	6.5	24	7 [14]	0 (0)	0
6.6-10.5	18	9 [1]	0 (0)	0	49	15 [2]	9 (3)	20	46	22 [3]	4.5 (4)	18	12	1 [9]	0 (0)	0
10.6-14.5	11	6 [4]	0 (0)	0	18	9 [6]	6 (2)	25	13	6 [1]	0 (0)	0	6	2 [0]	3 (1)	100
14.6-17.0	4	4 [0]	5 (1)	25	7	0 [0]	0 (0)	0	11	6 [2]	0 (0)	0	6	2 [1]	0 (0)	0
Total	119	20 [5]	10 (2)		193	35 [11]	17 (6)		301	88 [27]	9 (8)		170	33 [30]	3 (1)	

%a-percentage of total individuals with sinuses; %b-percentage of individuals with sinuses in each age group.

Harris Lines (1 and 2)

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b
0-0.5	25	9	2 (1)	11	10	0	0 (0)	0	45	15	0.7 (1)	7	40	22	27 (12)	54.5
0.6-2.5	36	12	11 (6)	50	45	10	12 (7)	70	70	29	7 (10)	34	82	22	29 (13)	59
2.6-6.5	25	13	16 (9)	69	64	16	24.5 (14)	87.5	85	48	20 (27)	56	24	1	2 (1)	100
6.6-10.5	18	11	12.5 (7)	63	49	20	23 (13)	65	46	34	9 (12)	35	12	-	-	-
10.6-14.5	11	7	7 (4)	57	18	8	7 (4)	50	13	6	0 (0)	0	6	-	-	-
14.6-17.0	4	4	3.5 (2)	50	7	3	2 (1)	33	11	5	0 (0)	0	6	-	-	-
Total	119	56	52 (29)		193	57	68 (39)		301	137	36 (50)		170	45	58 (26)	

%a-percentage of total individuals with X-rayed tibiae, %b-percentage of individuals with tibiae in each age group.

Harris Lines (2 only)

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b	number	with tibiae	%a (N)	%b
0-0.5	25	9	0 (0)	0	10	0	0 (0)	0	45	15	0.7 (1)	7	40	22	13 (6)	27
0.6-2.5	36	12	9 (5)	42	45	10	9 (5)	50	70	29	2 (3)	10	82	22	15.5 (7)	32
2.6-6.5	25	13	7 (4)	31	64	16	12 (7)	44	85	48	9 (12)	35	24	1	2 (1)	100
6.6-10.5	18	11	7 (4)	36	49	20	12 (7)	35	46	34	2 (3)	9	12	-	-	-
10.6-14.5	11	7	3.5 (2)	28	18	8	5 (3)	37.5	13	6	0	0	6	-	-	-
14.6-17.0	4	4	2 (1)	25	7	3	2 (1)	33	11	5	0	0	6	-	-	-
Total	119	56	28 (16)		193	57	41 (23)		301	137	14 (19)		170	45	31 (14)	

%a-percentage of total individuals with X-rayed tibiae, %b-percentage of individuals with tibiae in each age group.

Porotic Hyperostosis

	Raunds Furnells				St.Helen-on-the-Walls				Wharram Percy				Christ Church Spitalfields			
Age	number	with skull	%a (N)	%b	number	with skull	%a (N)	%b	number	with skull	%a (N)	%b	number	with skull	%a (N)	%b
0-0.5	25	22	0.9 (1)	4.5	10	4	0 (0)	0	45	36	2 (4)	11	40	20	2 (2)	10
0.6-2.5	36	29	2 (2)	7	45	23	2 (2)	9	70	61	2 (5)	8	82	56	5 (6)	11
2.6-6.5	25	24	0.9 (1)	4	64	28	0 (0)	0	85	73	0 (0)	0	24	22	0 (0)	0
6.6-10.5	18	16	0 (0)	0	49	23	1 (1)	4	46	29	0 (0)	0	12	12	0 (0)	0
10.6-14.5	11	11	0 (0)	0	18	12	0 (0)	0	13	10	0 (0)	0	6	3	0.8 (1)	33
14.6-17.0	4	4	0.9 (1)	25	7	0	0 (0)	0	11	8	0 (0)	0	6	2	0 (0)	0
Total	119	106	5 (5)		193	90	3 (3)		301	217	4 (9)		170	115	8 (9)	

%a-percentage of total individuals with a skull; %b-percentage of individuals with a skull in each age group.

St. Helen-on-the-Walls

Skeleton	*N lines**	*Tooth*	*Distance from CEJ (mm)*	*Crown height*	*Formation (yrs)*	*Rounded*
5085	2	C	7.55	12.19~	2.47	**2.5**
5189	2	C	0.97	12.19~	0.3	**0.5**
			5.67	12.19~	1.9	**2**
5252	2	C	2.40	12.19~	0.78	**1**
			5.09	12.19~	1.7	**1.5**
5510	2	C	6.30	12.19~	2.06	**2**
			9.59	12.19~	3.15	**3**
5513	3	C	3.01	12.19~	0.98	**1**
5526	2	C	1.90	12.19~	0.62	**0.5**
			3.56	12.19~	1.2	**1**
5599	1	C	4.18	11.98	1.39	**1.5**
5674	1	C	4.66	12.19~	1.5	**1.5**
5675	1	C	3.45	11.78	1.17	**1**
5691	1	C	4.23	12.19~	1.38	**1.5**
5707	2	C	3.83	11.37	1.34	**1.5**
5711	1	C	3.23	11.37	1.13	**1**
		Pm1	3.86	8.83	2.2	**2**
5793	1	C	2.52	10.28	0.98	**1**
5795	2	C	3.32	12.04	1.1	**1**
			4.54	12.04	1.5	**1.5**
5897	2	C	3.21	12.72	0.3	**0.5**
			5.36	12.72	1.7	**1.5**
		Pm1	3.66	9.32	2	**2**
			6.25	9.32	3.4	**3.5**
5904	3	C	1.94	12.87	0.6	**1**
			5.28	12.87	1.6	**1.5**
6004	2	C	3.40	13.01	1	**1**
			4.92	13.01	1.5	**1.5**
6092	3	C	1.16	11.35	0.4	**0.5**
			3.41	11.35	1.2	**1**
			5.13	11.35	1.8	**2**
		PM2	3.93	8.46	2.9	**3**
X	1.9					

** Maximum number of lines on a tooth.*

~ population mean crown height

Wharram Percy

Skeleton	*N lines**	*Tooth*	*Distance from CEJ (mm)*	*Crown height*	*Formation (yrs)*	*Rounded*
CN15	2	C	5.02	12.93	1.5	**1.5**
			7.40	12.93	2.3	**2.5**
		PM2	5.06	9.04	3.5	**3.5**
EE72	3	C	6.08	9.58	2.5	**2.5**
			3.26	9.58	1.4	**1.5**
		M1	2.90	6.75	0.9	**1**
			5.22	6.75	1.7	
EE81	1	C	5.97	12.46	1.92	**2**
EE102	1	C	4.20	11.53	1.45	**1.5**
G500	2	C	5.52	11.34	1.9	**2**
			7.04	11.34	2.5	**2.5**
G510	2	C	2.95	10.92	1.1	**1**
			5.34	10.92	1.9	**2**
G723	2	C	2.38	10.90	0.9	**1**
			5.39	10.90	2	**2**
NA2	1	C	3.61	11.57	1.2	**1**
		Pm1	2.61	7.58	1.7	**1.5**
NA19	2	C	4.65	13.93	1.3	**1.5**
NA30	2	M1	2.44	7.93	0.3	**0.5**
			4.32	7.93	1.2	**1**
NA98	2	C	2.66	10.92	2	**2**
			4.66	10.92	1.7	**1.5**
NA105	1	C	4.86	13.06	1.5	**1.5**
NA205	2	C	4.48	11.44~	1.6	**1.5**
			6.45	11.44~	2.25	**2**
V7	3	C	2.87	10.47	1.1	**1**
			5.16	10.47	1.9	**2**
			7.28	10.47	2.6	**2.5**
V12	3	C	4.11	12.79	1.3	**1.5**
			5.98	12.79	1.87	**2**
V47	1	C	5.37	11.11	1.9	**2**
WCO52	2	C	4.21	11.57	1.45	**1.5**
			7.78	11.57	2.7	**3**
WCO103	3	C	2.73	7.40	1.5	**1.5**
			4.46	7.40	2.4	**2.5**
			6.35	7.40	3.4	**3.5**
WCO134	3	C	1.51	11.44~	0.5	**0.5**
			3.91	11.44~	1.4	**1.5**
			6.83	11.44~	2.3	**2.5**
WCO202	2	M1	4.26	7.77	1.2	**1**
X	2					

** Maximum number of lines on a tooth.*

~ population mean crown height

Christ Church Spitalfields

Skeleton	*N lines**	*Tooth*	*Distance from CEJ (mm)*	*Crown height*	*Formation (yrs)*	*Rounded*
2235	2	M1	3.67	7.49	**1**	**1**
2866	1	M1	4.81	7.95	1.3	**1.5**
2135	1	C	2.66	11.15	0.9	**1**
2139	2	C	4.13	12.43	1.3	**1.5**
			2.32	12.43	0.7	**0.5**
2200	1	C	8.58	12.13	2.8	**3**
2199	3	C	2.09	11.63	0.7	**0.5**
			4.03	11.63	1.4	**1.5**
			5.59	11.63	1.9	**2**
2755	1	C	4.06	10.39	0.6	**0.5**
2662	1	C	2.70	11.00	1	**1**
		dc	4.83	6.64	0.6	**0.5**
2815	1	dc	3.68	6.97	0.4	**0.5**
X	1.4					

** Maximum number of lines on a tooth.*

~ population mean crown height

Appendix X: List of Pathologies

Raunds Furnells

Skeleton	*Age*	*Pathology*	*Classification/diagnosis*
5032	2.6-6.5	Lytic lesion, greater trochanter of left femur	TB, Infection, neoplasm
5059	14.6-17.0	Ankylosis of L4 and L5	Trauma
5130	6.6-10.5	Fractured clavicles	Trauma
5138	10.6-14.5	Rib periostitis	Lower respiratory infection
5177	NB-0.5	Woven bone on right clavicle	Non-specific infection? Trauma?
5200	10.6-14.5	Septic arthritis of left arm, atophy of right leg, hyperdontia	*cause not evident*
5240	0.6-2.5	Porotic hyperostosis and frayed rib fragment	Possible rickets, metabolic
5280	0.6-2.5	Rib periostitis	Lower respiratory infection
5297	0.6-2.5	Periostitis and pitting on mandible, maxilla and zygomatic	Possible scurvy, metabolic
5312	0.6-2.5	Pitted sphenoid, maxilla and new bone on maxillary foramen	Possible scurvy, metabolic
5317	6.6-10.5	Rib periostitis	Lower respiratory infection
5324	10.6-14.5	Osteochonditis dissecans on tibial epiphyses	Circulatory
5335	2.6-6.5	Rib periostitis; woven bone on left first metatarsal	Non-specific infection
5338	6.6-10.5	Rib Periostitis	Lower respiratory infection
5348	10.6-14.5	New bone on lumbar and sacral vertebrae, lytic lesion ilium	Gastrointestinal TB
5352	10.6-14.5	Paralysis of left leg, ectocranial new bone	*cause not evident*

St. Helen-on-the-Walls

Skeleton	*Age*	*Pathology*	*Classification/diagnosis*
5077	2.6-6.5	Exostosis on proximal humerus	Developmental?
5085	6.6-10.5	Ulceration on right parietal	Ringworm? Infection
5092	39 weeks	Bifid rib	Congenital
5108	14.6-17.0	Fractured tibia, woven bone on tibia and femur	Trauma, infection
5189	10.6-14.5	Depressed fracture on right parietal	Trauma
5271	6.6-10.5	Woven bone on right clavicle	Non-specific infection
5272a	0.6-2.5	Patch of woven bone on right femur	Non-specific infection
5340b	0.6-2.5	Woven bone on right temporal bone	Non-specific infection
5470	2.6-6.5	Bowed left tibia and fibula, frayed distal surfaces	Rickets, metabolic
5578	?	Lytic destruction of left auricular surface	Gastrointestinal TB
5707	6.6-10.5	Rib periostitis	Lower respiratory infection
5764	10.6-14.5	Blastic and lytic lesions on sacral and lumbar vertebrae	?TB, infection
5814	10.6-14.5	New bone on left ulna and fibula	Non-specific infection
5822	6.6-10.5	Oblique fracture of mandible	Trauma
5917	6.6-10.5	Haematoma on left tibia	Trauma
5947	2.6-6.5	Endocranial and ectocranial new bone, orbits	Scurvy, metabolic
6004	10.6-14.5	New bone on tibiae, fibulae, femora and radii	Scurvy, metabolic

Skeleton	*Age*	*Pathology*	*Classification/diagnosis*
EE29	14.6-17.0	Irregular new bone on sacrum (S1-3)	TB, fungal infection.
G304	2.6-6.5	Depressed fracture on the skull.	Perimortem trauma
G308	0.6-2.5	Porotic hyperostosis of skull	Rickets/anaemia, metabolic
G534	NB-0.5	Porotic hyperostosis of skull	Rickets/anaemia, metabolic
G658	6.6-10.5	Rib periostitis, new bone on right tibia	Lower respiratory. infection
G708	6.6-10.5	New bone on PPMN	Rhinitis, infection
NA19	6.6-10.5	Healed depressed fracture: skull	Trauma
NA48	2.6-6.5	Inflammatory pitting on PPMN	Rhinitis, infection
NA65	2.6-6.5	Left leg one inch shorter than right	*cause not evident*
NA92	NB-0.5	Porotic changes to skull	Early rickets? Metabolic
NA93	NB-0.5	Porosity of long bones	Rickets/scurvy, metabolic
NA98	2.6-6.5	Disuse atrophy of left leg and foot	*cause not evident*
NA103	2.6-6.5	Rib lesions	Lower respiratory infection
NA108	0.6-6.5	Changes to skull and rib ends	Rickets/scurvy/anaemia
NA118	0.6-2.5	New bone above both external auditory meati.	Non-specific infection
NA131	?	Flared end of radius, skull lesions	Rickets, metabolic
NA135	2.6-6.5	Lytic destruction of mandible	Neoplasm
NA139	NB-0.5	Beading of rib ends?	Rickets? Metabolic
NA149a	NB-0.5	Fractures at epiphyseal ends of tibia, femur, radius	Early sign rickets/ trauma
NA160	0.6-2.5	New bone on tibia and scapula	Scurvy/ICH
NA164	0.6-2.5	New bone on occipital (ectocranial)	Non-specific infection
NA174	6.6-10.5	New bone on left femur	Non-specific infection
NA178	14.6-17.0	Healed depressed fracture on skull	Trauma
NA189	0.6-2.5	Vascular rib ends and long bones	Healed rickets? Metabolic
NA194	0.6-2.5	Changes to skull, orbits, ribs	Rickets/scurvy/anaemia
NA191	NB-0.5	Vascular rib ends and longbones	Healed rickets? Metabolic

PPMN: Nasal surface of the maxillary palatine process

ICH = Infantile Cortical Hyperostosis

Wharram Percy continued...

NA225	0.6-2.5	New bone on sphenoid, frontal, orbits	Scurvy? Metabolic
NA236	0.6-2.5	Porotic hyperostosis	Anaemia, metabolic
SA55	0.6-2.5	New bone on scapula and humerus	Scurvy? Trauma/ICH
V35	6.6-10.5	New bone on sacrum and ilia	Tuberculosis, fungal infection?
V24	14.6-17.0	Healed fracture of right clavicle	Trauma
V28	0.6-2.5	Skull changes at fontanelle and sternal rib ends	Rickets/scurvy/ICH?
V55	?	Blastic lesion on parietal	Ulcer/ ringworm? Infection
V57	NB-0.5	Flared end of radius	Rickets? Metabolic
WCO39	14.6-17.0	Isolated lesion on proximal tibia	Neoplasm, cyst, infection?
WCO42	NB-0.5	Hyperostosis of skull	Rickets? Metabolic
WCO58	6.6-10.5	New bone ribs, humeri, pelvis, scap, clavs	Lower respiratory.infection/TB/ICH?
WCO69	10.6-14.5	Superior slipped femoral epiphysis	Trauma
WCO99	6.6-10.5	New bone on frontal and orbital crests	Infection/Trauma
WCO107	?	New bone on ribs and ischium	Lower respiratory infection

Christ Church Spitalfields

Skeleton	*Age*	*Pathology*	*Classification/diagnosis*
2128	2.6-6.5	Necrosis of left olecranon; atrophy of left arm	?Trauma
2147	NB-0.5	Frayed epip. at wrist, knee, ankles, beading of ribs	Rickets/scurvy, metabolic
2148	0.6-2.5	Frayed epip. at wrist, knee, ankles, beading of ribs, collapsed distal tibia, bowed/flattened fibula	Rickets, metabolic
2199	2.6-6.5	Bowed humeri, beaded ribs, flaring of wrists, knees and elbows suggest rickets at crawling age.	Rickets, metabolic
2206	0.6-2.5	Porotic hyperostosis, beaded ribs, bowed tibs/ fibs	Rickets, metabolic
2235	6.6-10.5	Severe hypoplasias	Non-specific stress
2239	10.6-14.5	Osteitis of left tibia, periostitis right MT5 and MC2	Non-specific infection
2261	0.6-2.5	Beaded ribs, frayed and flaring at knees, wrists, ankles	Rickets, metabolic
2374	?	Perimortem cut on left humerus	Autopsy
2412	0.6-2.5	Perimortem cut to frontal	Autopsy
2429	0.6-2.5	Beaded ribs, medial bowing of fibulae	Rickets, metabolic
2440	0.6-2.5	Beaded ribs, thickened flat and long bones, curving of ilia	Rickets, metabolic
2441	0.6-2.5	Flattened/bowed fibula, slipped femoral epiphyses	Rickets/trauma
2455	0.6-2.5	Perimortem cuts to frontal bone	Autopsy
2512	NB-0.5	Porotic hyperostosis, new bone on maxilla, beaded ribs	Rickets/scurvy, metabolic
2532	0.6-2.5	Beaded/bowed ribs, thick flat bones, wrist severely affected	Rickets, metabolic
2559	0.6-2.5	Beaded ribs, joints frayed and flared	Rickets, metabolic
2621	2.6-6.5	Rib periostitis; woven bone on tibiae	Non-specific infection
2626	0.6-2.5	Flaring of femora, distal medial bowing of tibiae	Rickets, metabolic
2637	0.6-2.5	Severe hypoplasias, bowed tibs, femora, ulna, trabecular bone	Rickets, metabolic
2657	NB-0.5	Fractured clavicle	Trauma
2662	6.6-10.5	Bowed/shortened left femur; short left. Tibia and fibula	?Trauma/healed rickets
2691	0.6-2.5	Pott's disease; TB meningitis (?), beaded ribs	Rickets and tuberculosis
2692	2.6-6.5	Destruction and new bone on pelvis, C4-7 lytic collapse, lytic lesion of right knee, Psoas abscess of acetabulum (?)	Tuberculosis, infection

Christ Church Spitalfields continued...

2700	14.6-17.0	Fracture of 11th rib	Trauma
2724	0.6-2.5	Ribs, knee, wrists frayed	Early rickets, metabolic
2726	0.6-2.5	Beading ribs and thick long bones, diffuse perios.	Rickets/scurvy, metabolic
2734	0.6-2.5	Malformed humeral head, flared and frayed joints	Rickets, metabolic
2755	10.6-14.5	Fractured clavicle, humeral head and epiphysis, right arm 1cm shorter than left	Trauma
2763	10.6-14.5	Beading of ribs, fraying of distal tibia	Rickets, metabolic
2772	0.6-2.5	Porotic hyperostosis, all joints flared and frayed	Rickets, metabolic
2774	0.6-2.5	Fractured clavicle, bowed long bones, thickened flat bones, thinned cortex, beaded ribs	Trauma/Rickets
2775	NB-0.5	Beaded ribs, flared epiphyses, thinned cortices	Rickets, metabolic
2792	NB-0.5	3 fractured ribs, woven bone at break (recent)	Trauma
2841	0.6-2.5	Bowed tibiae, frayed/flared epip. thick flat bones	Rickets, metabolic
2874	NB-0.5	Beaded ribs, frayed but not flared epiphyses	Rickets/scurvy, metabolic
2876	0.6-2.5	Bowed tibiae, beaded ribs, flared ankles and knees	Rickets, metabolic
2877	0.6-2.5	Beaded ribs, frayed and flared tibiae and femora	Rickets, metabolic
2898	?	Beaded ribs, porotic hyper. frayed/flared epiphyses	Rickets, metabolic
2880	NB-0.5	Beaded ribs, flared/frayed knees, ankles and wrists	Rickets, metabolic

Appendix XI: Summary of Sample Representation and Ageing Methods

Table A Number of non-adults in the present study compared to previous reports.

Site	*Author*	*No*	*Number in present study*	*Number unaccounted for*
Raunds Furnells	Powell (1996)	170	149	21
St. Helen	Dawes and Magilton (1980)	317	231	86
Wharram Percy	unpublished site	-	303	-
Spitalfields	Molleson and Cox	215	206	9
TOTAL			889	

Table B Number of individuals aged by various methods

Site	*Method*						TOTAL
	Dental development		Long bone length		Bone maturation		
	Moorrees *et al.* (1963ab)	Ubelaker (1978)	Scheuer *et al.* (1980)	Ubelaker (1978)	Epiphysel fusion	Tympanic ring	
Raunds Furnells	98	1	36	5	0	2	142
St. Helen	100	6	15	77	1	0	200
Wharram Percy	153	16	62	64	4	4	303
Spitalfields	116	0	43	22	1	4	186
TOTAL	467	23	156	168	6	10	830 in aged sample